

NATIONAL CENTER FOR PROSECUTION OF CHILD ABUSE

Special Topics in Child Abuse

Overcoming Defense Expert Testimony in Abusive Head Trauma Cases

By Dermot Garrett

Edited by Eleanor Odom,

Amanda Appelbaum

and David Pendle



Scott Burns

Director, National District Attorneys Association

The National District Attorneys Association is the oldest and largest professional organization representing criminal prosecutors in the world. Its members come from the offices of district attorneys, state's attorneys, attorneys general, and county and city prosecutors with responsibility for prosecuting criminal violations in every state and territory of the United States. To accomplish this mission, NDAA serves as a nationwide, interdisciplinary resource center for training, research, technical assistance, and publications reflecting the highest standards and cutting-edge practices of the prosecutorial profession.

In 1985, the National District Attorneys Association recognized the unique challenges of crimes involving child victims and established the National Center for Prosecution of Child Abuse (NCPCA). NCPCA's mission is to reduce the number of children victimized and exploited by assisting prosecutors and allied professionals laboring on behalf of victims too small, scared or weak to protect themselves.

Suzanna Tiapula

Director, National Center for Prosecution of Child Abuse

A program of the National District Attorneys Association

www.ndaa.org

703.549.9222

This project was supported by Grants #2010-CI-FX-K008 and [new VOCA grant #] awarded by the Office of Juvenile Justice and Delinquency Prevention. The Office of Juvenile Justice and Delinquency Prevention is a component of the Office of Justice Programs. Points of view in this document are those of the author and do not necessarily represent the official position or policies of the U.S. Department of Justice.

Overcoming Defense Expert Testimony in Abusive Head Trauma Cases

BY DERMOT GARRETT

Former Staff Attorney, NDAA's National Center for PCA, Currently Assistant
State's Attorney, Montgomery County (Maryland) State's Attorney's Office

EDITED BY

Eleanor Odom, *NCPCA Fellow*

Amanda Appelbaum, *NCPCA Staff Attorney*

David Pendle, *NCPCA Staff Attorney*

Page 2: **Blank**

TABLE OF CONTENTS

5
Introduction

6
Pretrial Defense Claim:
The evidence that children can sustain brain injuries from having been shaken
is too unreliable to be admitted in court under *Daubert*, *Frye*, or state-specific admissibility tests.

8
Defense Claim:
It is biomechanically impossible to cause massive brain injuries including
subdural hematomas in children through shaking alone.

16
Defense Claim:
It is impossible to cause brain injuries though shaking absent a concomitant neck injury.

19
Defense claim:
Short falls commonly produce the types of fatal cranial injuries seen in AHT cases.

23
Defense Claim:
A hypoxic rather than traumatic event might cause subdural hemorrhage.

25
Defense Claim:
A child's previously existing subdural hematoma may "rebleed," either spontaneously
or as a result of a trivial injury, and cause severe brain injuries.

29
Defense Claim:
The infant may have suffered a head injury without manifesting symptoms
until hours or days later—a "lucid interval."

32
Defense Claim:
"The best study, of which I'm aware, says that 75 percent will occur within the first 24 hours,
and the remaining 25 percent can take up to three or four days."

35
Defense Claim:
Retinal hemorrhages are nonspecific injuries and do not indicate child abuse.

42
Conclusion

Page 4: **Blank**

INTRODUCTION

“ABUSIVE HEAD TRAUMA” (AHT) is the recommended medical diagnosis to describe the constellation of injuries resulting from the intentional infliction of head trauma, including those injuries traditionally ascribed to “Shaken Baby Syndrome.”¹ AHT cases involve a number of challenges, including presenting complex medical information to a jury. Another challenge stems from a group of physicians who testify frequently and convincingly for the defense in AHT cases, even though many of their opinions are outside the consensus of the medical community.² In order to effectively cross-examine the defense experts and communicate accurate information to the jury, it is essential for prosecutors to have a basic understanding of the medical issues likely to be raised by the defense in AHT cases.

Each section of this article examines a claim frequently raised by the defense in AHT cases. The transcript quotes are examples of defense testimony and all of these transcripts and materials are available through NDAA’s National Center for Prosecution of Child Abuse (NCPCA).

¹ Cindy W. Christian, Robert Block and the Committee on Child Abuse and Neglect, *Abusive Head Trauma in Infants and Children*, 123 Pediatrics 1409, 1411 (2009).

² *In re Child of Green*, Nos. C3-03-125, C1-03-205, 2003 WL 21652472 at *3 (Minn. Ct. App. 2003) (“the district court found his testimony to be not credible because Dr. Plunkett...has a theory regarding sudden-impact injuries that ‘is not generally accepted in the relevant medical/scientific community’”); *A Local Authority v S* [2009] EWHC 2115 (Fam) (This article can be viewed at: <http://www.familylawweek.co.uk/site.aspx?i=ed53850>). “Dr. Squier and Dr. Cohen, I find with regret, have each fallen into that category of expert identified . . . who has developed a scientific prejudice. As a consequence . . . Dr. Squier has permitted her convictions to lead her analysis. . . each of the significant factual errors made by her served to support her hypothesis of choking and hypoxia.”

Pretrial Defense Claim:

**The evidence that children can sustain brain injuries
from having been shaken is too unreliable to be admitted in court
under *Daubert*, *Frye*, or state-specific admissibility tests.**

Defense attorneys will often file pretrial motions to preclude the state from offering testimony regarding “shaken baby syndrome” or testimony that children’s brains can be injured through acceleration and deceleration forces.³ The motion may be based on the untrue claim that biomechanical research has falsified many of the scientific tenants underlying the AHT diagnosis.

Accurate Evidence: *Every jurisdiction that has considered the issue currently holds that AHT evidence is admissible under Daubert, Frye, or state-specific admissibility tests.*⁴

The defense will often cite a single 2006 Kentucky trial court decision excluding AHT evidence under *Daubert*,⁵ *Commonwealth v. Davis*, without mentioning that the decision was subsequently overturned in 2008.⁶ In *Davis*, the trial judge excluded the testimony regarding “shaken baby syndrome,” holding that the state’s proffered evidence supporting the existence of the syndrome was too dependent upon clinical rather than “scientific” studies. The Kentucky Court of Appeals overturned the trial court decision, finding that clinical studies are not inherently unreliable and are appropriate given the impossibility of employing the scientific method with infants in AHT studies. Furthermore, cross-examination is the appropriate means of attacking weaknesses in expert testimony, with the jury deciding the relative merit of the testimony.

Defense pretrial briefs may misleadingly cite a 2008 Wisconsin appellate decision, *State v. Edmunds*,⁷ for the notion that AHT evidence is no longer considered scientifically reliable.

³ Sample motions available through the NCPCA.

⁴ **Frye Cases:** *People v. Leslie Martin*, (Bronx County 2008); *In re Child of Green*, Nos. C3-03-125, C1-03-205, 2003 WL 21652472, at *2 (Minn. Ct. App. 2003). (Frye/Mack test); *State v. McClary*, 541 A.2d 96, 102 (Conn. 1988); *People v. Ceasor*, 2007 WL 2011747, at *2 (Mich. App. 6 Dist. 2007)(Frye/Reed test); *Illinois v. Armstrong*, 395 Ill. App. 3d 606 (Ill. App. Ct. 2009); *Commonwealth v. Passarelli*, 2001 PA Super. 377, 789 A.2d 708, 729 (Pa. Super. 2001); *Herliby v. State*, 927 So. 2d 146 (Fla. Dist. Ct. App. 2006). **Daubert Cases:** *Commonwealth v. Davis*, 2008 Ky. App. LEXIS 186 (Ky. Ct. App. 2008); *State v. Leibhart*, 662 N.W.2d 618, 628 (Neb. 2003); *State v. Vandemark*, 2004 Del. Super. LEXIS 376 (Del. Super. Ct. 2004); *State v. Carr*, 2010 Ohio 2764, 28 (Ohio Ct. App., Hamilton County June 18, 2010); **State Admissibility Tests:** *People v. Renteria*, 2005 Cal. App. Unpub. LEXIS 8834 (Cal. Ct. App. 2005)(unpublished opinion); *People v. Cauley*, 32 P.3d 602 (Co. Ct. App. 2001); *State v. Lopez*, 412 S.E.2d 390, 393 (S.C. 1991); *Utah v. Mendoza* (2008).

⁵ *Commonwealth v. Davis*, No. 2006-CA-002237 (Ky. Ct. App. 2006).

⁶ *Commonwealth v. Martin*, 2008 Ky. App. LEXIS 186 (Ky. Ct. App. 2008).

⁷ *State v. Edmunds*, 746 N.W.2d 590 (Wis. Ct. App. 2008).

The *Edmunds* court overturned a woman's 1997 conviction for killing her child through abusive head trauma. The Wisconsin appellate court was persuaded by expert testimony that there was a "reasonable probability" that a jury in 2008 might come to a different decision based on emerging science.⁸ However, *Edmunds* involved a post-trial *habeas* petition for relief rather than a pre-trial motion *in limine* to exclude evidence, and is inapplicable for the purposes of *Frye* or *Daubert* admissibility tests.⁹ The focus of the *Edmunds* court was on "new evidence," specifically whether medical testimony available in 2008 that was not available in 1997 would be likely to change the verdict. *Edmunds* did not hold that AHT evidence was inadmissible, but rather that the jury might have found differently given the "new evidence" available.

Defense motions may cite biased law review articles as authoritative sources, when in fact those articles omit science that fails to support the defense position. For instance, defense motions may cite an article by Gene Lyons, written as a third-year law student, entitled *Shaken Baby Syndrome: A Questionable Scientific Syndrome and a Dangerous Legal Concept*.¹⁰ The Lyons article has been criticized for presenting a biased argument that excludes "over 500 articles that support the validity of the shaken baby syndrome."¹¹ Lyons inaccurately claims that "one of the main diagnostic findings leading to an SBS diagnosis is the absence of any other signs that abuse has occurred."¹² In fact, although scalp injuries, skull fractures, rib fractures and metaphyseal leg fractures frequently co-occur with AHT, the diagnosis can be made either in conjunction with or in the absence of other signs of abuse. For an in-depth analysis of the Lyons article, see the State's Memorandum in Opposition to Motion to Exclude, *Utah v. Mendoza*,¹³ available through the NCPCA.

Defense motions may also mischaracterize the state's argument, and then argue against a straw man. In *Georgia v. Noel*, the defense sought to exclude mention of "5 Alleged Facts" that supposedly were evidence of shaking injuries, including that "retinal hemorrhages are diagnostic of non-accidental trauma."¹⁴ The defense statement oversimplifies and distorts the state's

⁸ *Id.* at 599 ("There is a reasonable probability that a jury, looking at both the new medical testimony and the old medical testimony, would have a reasonable doubt as to Edmunds's guilt.")

⁹ *Id.*

¹⁰ Gene Lyons, Note, *Shaken Baby Syndrome: A Questionable Scientific Syndrome and a Dangerous Legal Concept*, 2003 Utah L. Rev. 1109 (Summer 2003).

¹¹ Memorandum in Opposition to Motion to Exclude, *Utah v. Mendoza*, (Utah Dist. Ct. 2008).

¹² Gene Lyons, Note, *Shaken Baby Syndrome: A Questionable Scientific Syndrome and a Dangerous Legal Concept*, 2003 Utah L. Rev. 1109, 1126 (Summer 2003).

¹³ Memorandum in Opposition to Motion to Exclude, *Utah v. Mendoza*, (Utah Dist. Ct. 2008).

¹⁴ Motion in Limine, *State v. Noel*, No. 07-SC-55406 (Ga. Super. Ct. Aug. 13, 2009).

position. Retinal hemorrhages in children, although highly associated with non-accidental injury, can also result from a number of non-inflicted causes,¹⁵ and are neither necessary nor dispositive for an AHT diagnosis. In *Noel*, the defense motion states that “[s]tudies have shown that accidental trauma causes retinal hemorrhage as often as non-accidental trauma.”¹⁶ The article cited does not support that argument, as should be clear from the article title: *Systemic and Ocular Findings in 169 Prospectively Studied Child Deaths: Retinal Hemorrhages Usually Mean Child Abuse*.¹⁷ The association between retinal hemorrhages and non-accidental head injuries is explored more thoroughly later in this article.

Many of the articles the defense accurately cites are written by other defense experts. John Plunkett’s 2001 study is frequently cited for the proposition that short falls can produce severe or fatal injuries with symptoms that mimic those associated with abusive head trauma.¹⁸ To counter Plunkett’s article, the prosecutor can draw attention to the author’s position as a frequent defense expert. Regarding Plunkett’s credibility, the Minnesota Court of Appeals noted that “the district court found his testimony to be not credible because Dr. Plunkett...has a theory regarding sudden-impact injuries that ‘is not generally accepted in the relevant medical/scientific community.’”¹⁹ Please see the “Short Falls” and “Lucid Interval” sections of this article for further discussion of the Plunkett study.

Defense Claim:
It is biomechanically impossible
to cause massive brain injuries including
subdural hematomas in children
through shaking alone.

The defense expert may testify that a person cannot shake a baby hard enough to produce bleeding between the dura and the arachnoid membranes, known as a subdural hematoma (SDH).

¹⁵ Henry F. Krous & Roger W. Byard, *Controversies in Pediatric Forensic Pathology*, 1 Forensic Sci. Med. Pathology. 9, 13 (2005).

¹⁶ Motion in Limine, *State v. Noel*, No. 07-SC-55406 (Ga. Super. Ct. Aug. 13, 2009).

¹⁷ “In the absence of a verifiable history of a severe head injury or life-threatening central nervous system disease, retinal and ocular hemorrhages were diagnostic of child abuse.” M.G.F. Gilliland et al., *Systemic and Ocular Findings in 169 Prospectively Studied Child Deaths: Retinal Hemorrhages Usually Mean Child Abuse*, 68 Forensic Sci Intl 117-132 (1994).

¹⁸ John Plunkett, *Fatal Pediatric Head Injuries Caused by Short-Distance Falls*, 22 Am. J. Forensic Med. Path. 1 (2001).

¹⁹ *In re Child of Green*, Nos. C3-03-125, C1-03-205, 2003 WL 21652472, at *3 (Minn. Ct. App. 2003).

Two examples of such testimony are below:

- “[H]uman beings can’t shake children hard enough to cross those injury threshold ... you’re not going to give a baby an acute subdural hematoma by shaking it.”²⁰
- “I am not saying that children can’t get rotational injuries to the brain, I’m just saying it can’t happen from shaking.”²¹

Before addressing the biomechanics argument, it is important to emphasize that the data regarding strain and shear tolerances in children is entirely approximative. One cannot design an experiment that involves shaking infants to determine injury thresholds for the subdural membrane, cortical bridging veins, cervical spine, etc. Absent this ability to employ the scientific method, scientists and doctors have relied on mass scaling to extrapolate from animals to human infants. A 2010 study by Ibrahim demonstrated that simple mass scaling is ineffective, and that other factors such as age-related biochemical differences influence the body’s reaction to injury.²² Clinical studies that evaluate infants directly, such as the confession literature and the studies described in the “short falls,” “lucid interval,” and “rebleed” sections of this article, indicate that an infant can suffer cerebral injuries through shaking. Biomechanical and animal studies, while a valuable adjunct to clinical studies, must be evaluated in light of the limitations of utilizing mass scaling and approximative data.

SDH, while not indicative of abuse when found in isolation, is virtually diagnostic for abuse when found in conjunction with severe retinal hemorrhage (RH) and absent signs of impact.²³ In 2009, Vinchon studied 45 cases of confessed inflicted head injury (IHI) and 39 cases of witnessed accidental trauma (AT) to determine which clinical, radiological, and ophthalmological variables were most correlated with inflicted versus accidental head trauma.²⁴ Severe retinal hemorrhage (RH), the absence of signs of impact, and SDH were the features most

²⁰ Testimony of Dr. Ronald Uscinski at 63-64, *State v. Listro* No. TTD-CR08-0092447-T(Conn. Super. Ct. March 12, 2010).

²¹ Transcript of Janice Ophoven, *State v. Helms*, No. RIF – 102020 (Cal. App. Dep’t Super. Ct., March 4, 2004).

²² N. Ibrahim, J. Ralston, C. Smith, S. Margulies, *Physiological and pathological responses to head rotations in toddler piglets*, 27 J. Neurotrauma, 1021-35 (2010).

²³ Vinchon M, de Foort-Dhellemmes S, Desurmont M, Delestret I. *Confessed abuse versus witnessed accidents in infants: comparison of clinical, radiological, and ophthalmological data in corroborated cases*. Childs Nerv Syst. 2010 May; 26(5):637-45. Epub 2009 Nov 28.

²⁴ *Id.*

highly correlated with IHI.²⁵ When all three features were present, the predictive value for IHI was 100 percent.²⁶ In Vinchon's study, seven of ten fatal IHI cases had signs of impact, while three fatal cases had no signs of impact.²⁷ Vinchon concluded that "shaken impact baby syndrome represents a subgroup of IHI with a worse prognosis but that SBS without impact can also be fatal."²⁸

The defense expert will often cite a 1987 study by Duhaime²⁹ as proof that human beings cannot shake a child hard enough to produce brain injuries such as subdural hematomas. Duhaime measured accelerational forces generated when people shook models of one-month-old infants. The models were crudely constructed with either a metal hinge neck, a low-resistance rubberneck, or a moderate-resistance rubberneck.³⁰ Duhaime found that shaking alone did not generate enough force to reach injury thresholds for concussion, subdural hemorrhage, or diffuse axonal injury in any of the models she constructed.³¹ When impact was added to shaking, however, the forces generated exceeded all three injury thresholds. Duhaime concluded that shaking without impact was "unlikely to cause the shaken baby syndrome."³²

Accurate Science: *Duhaime's experiment did not accurately measure the amount of force caused by shaking an infant because she used dummies with necks that did not mechanically resemble an infant's neck.*

Duhaime did not take into account any response characteristics of infants in building her crude models.³³ In 2003, Cory and Jones sought to test the validity of Duhaime's results by determining whether the type of model used affected the amount of force generated through shaking.³⁴ Cory and Jones found that varying the materials and construction of the models produced different angular accelerations. Three factors specifically increased head acceleration: the metal hinge-neck, a high center of gravity, and a cotton wool chest and back.³⁵ Models constructed

²⁵ *Id.* at 637.

²⁶ *Id.* at 643.

²⁷ *Id.*

²⁸ *Id.*

²⁹ Ann-Christine Duhaime, et al., *The Shaken Baby Syndrome: A Clinical, Pathological, and Biomechanical Study*, 66 J. Neurosurgery 409-415 (1987).

³⁰ *Id.* at 412.

³¹ *Id.* at 409.

³² *Id.*

³³ Cory, C.Z. & Jones, M.D., *Can Shaking Alone Cause Fatal Brain Injury? — A Biomechanical Assessment of the Duhaime Shaken Baby Syndrome Model*, 43 Med. Sci. L. 317, 317 (2003).

³⁴ *Id.* at 321.

³⁵ *Id.* at 323.

with those three characteristics, when subjected to an “increased shake effort and altered shake pattern,” achieved acceleration levels that exceeded those found in the Duhaime study.³⁶ The results from Cory and Jones’s study “emphasize a requirement that models for the investigation of SBS simulate an infant as accurately as possible in terms of mass distribution (centre of gravity) and response (biofidelity) of neck, chest and back.”³⁷ The Cory and Jones study illustrates the limitations of the models used by Duhaime. Furthermore, the results from the Cory and Jones study invalidate the theory frequently offered by defense experts that Duhaime’s study proves that shaking alone cannot cause severe head injury in infants. Even Duhaime herself has attempted to clarify her position stating “that the researchers ‘never said that beyond a shadow of a doubt shaking cannot cause injuries.’ What they said was that ‘shaking at least with this model, produces angular decelerations which are too small to cause the target injuries for which there are established thresholds.’”³⁸

Accurate Science: *Cory and Jones found that using injury thresholds based upon impact data may be necessary when calculating the forces generated by shaking, but Duhaime calculated her results using injury thresholds based upon angular acceleration data only.*

Duhaime used thresholds derived from a series of primate experiments³⁹ that purposely prevented the possibility of the head striking the chest and back during the whiplash motion.⁴⁰ Cory and Jones’s study showed that, with some models, shaking caused both chin to chest impacts as well as occiput to back impacts.⁴¹ Duhaime, however, used tolerance limits based only on angular acceleration data, not impact data.⁴² When Cory and Jones used an impact-based tolerance limit (Head Injury Criterion (HIC)) to assess their results, they found that 80 percent were over 840, the critical loading value suggested for children. A critical loading value was defined as “the load on the body under which an initial considerable damage of the organism takes

³⁶ *Id.* at 329.

³⁷ *Id.* at 329.

³⁸ 319 quoting Ann-Christine. Duhaime, *Research on the pathophysiology of the shaking-impact syndrome*.

³⁹ Cory, C.Z. & Jones, M.D., *Can Shaking Alone Cause Fatal Brain Injury? — A Biomechanical Assessment of the Duhaime Shaken Baby Syndrome Model*, 43 Med. Sci. L. 317, 327 (2003); Gennarelli, T.A., Thibault, L.E. & Adams J.H., et al., *Diffuse axonal injury and traumatic coma in the primate*, 12 Annals Neurosurgery 564-574 (1982).

⁴⁰ Cory, C.Z. & Jones, M.D., *Can Shaking Alone Cause Fatal Brain Injury? — A Biomechanical Assessment of the Duhaime Shaken Baby Syndrome Model*, 43 Med. Sci. L. 317, 327 (2003)

⁴¹ *Id.* at 325.

⁴² *Id.* at 327.

place—destruction of a cell; irreversible injury—for instance when bone fractures occur or primary organs rupture.”⁴³ These results indicate “another injury mechanism in ‘pure shaking,’ not previously investigated,”⁴⁴ and thus Cory and Jones concluded that the tolerance limits utilized by Duhaime “may not be applicable in the assessment of shaking simulations due to the impacts identified in [their] study.”⁴⁵

Accurate Science: *Duhaime derived injury thresholds from studies in which primates were exposed to a single angular acceleration-deceleration force, which fails to account for the cumulative damage caused by repeated shaking incidents.*⁴⁶

In determining injury thresholds, Duhaime relied in part on a study in which primates were exposed to a single acceleration-deceleration force.⁴⁷ Duhaime did not account for the cumulative damage caused when a child is repeatedly shaken. A 2010 study of defendant confessions revealed that shaking was described as extremely violent and was repeated in 55 percent of cases (between two and 30 times).⁴⁸ Animal experiments show that repeated injury to the brain has a cumulative effect in terms of causing damage.⁴⁹ Raghupathi, et al.’s 2004 study on piglets demonstrated that a second incident of rotational forces produced brain injury at a lower level of force than the first incident of rotational forces.⁵⁰ Thus the injury thresholds in Duhaime’s experiments may have been too high because, among other reasons, Duhaime did not consider that injury may occur at lower accelerational levels after repeated shaking incidents.

Accurate Science: *Duhaime used simple mass-scaling in extrapolating injury thresholds from an adult primate’s brain to a human infant’s brain, and simple brain mass scaling does not accurately predict thresholds for traumatic axonal injury in immature brains.*⁵¹

⁴³ Sturtz G., *Biomechanical data of children*. 24th Stapp Car Crash Conf. Proc., SAE. Paper No. 801313 (1980).

⁴⁴ Cory, C.Z. & Jones, M.D., *Can Shaking Alone Cause Fatal Brain Injury?—A Biomechanical Assessment of the Duhaime Shaken Baby Syndrome Model*, 43 Med. Sci. L. 317, 329 (2003).

⁴⁵ *Id.* at 331.

⁴⁶ Catherine Adamsbaum et al., *Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking*, 126 Pediatrics 546 (2010).

⁴⁷ Ramesh Raghupathi, et al., *Traumatic Axonal Injury is Exacerbated following Repetitive Closed Head Injury in the Neonatal Pig*, 21 J. Neurotrauma 307, 310 (2004).

⁴⁸ Catherine Adamsbaum et al., *Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking*, 126 Pediatrics 546 (2010).

⁴⁹ Ramesh Raghupathi, et al., *Traumatic Axonal Injury is Exacerbated following Repetitive Closed Head Injury in the Neonatal Pig*, 21 J. Neurotrauma, 307, 310 (2004).

⁵⁰ *Id.*

⁵¹ Margulies SS and Spivack BS. *Pathobiology and Biomechanics of Inflicted Childhood Neurotrauma in Inflicted Childhood Neurotrauma*, American Academy of Pediatrics, 2003. RM Reece and CE Nicholson eds. pp. 221-236.

Duhaime relied upon injury tolerance levels scaled from adult primates to adult humans, and then further scaled from adult humans to infants.⁵² However there are significant differences in how the human toddler and infant brain respond to injury, beyond what would be expected through simple mass scaling.⁵³ There are also significant and obvious differences between a monkey and a human brain. Defense experts may attempt to minimize the difficulty of extrapolating from an adult monkey's brain to a human infant's brain. The exchange below took place between the prosecutor and defense witness Dr. Ronald Uscinski in the 2010 case of *State v. Listro*:⁵⁴

Q: You would agree with me that the primate brain is different than the adult brain?

A: How so?

Q: You wouldn't agree with me?

A: Mass is mass. You take one cubic centimeter of the brain of a squirrel monkey, a chimpanzee, or a human being. They're gonna be the same mass. . . mass is mass and mass is the key thing.

Despite Dr. Uscinski's claims, recent literature shows that mass scaling is ineffective within the human species alone, much less between humans and monkeys. In comparison to human toddlers, infant brains are better able to resist impact injuries,⁵⁵ but may be more susceptible to shearing injury,⁵⁶ which is why toddlers who sustain a closed head injury rarely suffer the degree of diffuse brain atrophy seen in infants.⁵⁷ In a 2010 study, Ibrahim and Margulies compared the tissue vulnerability of infant and toddler piglets, and determined that "the toddler brain can withstand over three times greater strains than the infant before axonal injury results."⁵⁸ The authors conclude that "the traditional mechanical engineering approach of scaling by brain mass and stiffness cannot explain the vulnerability of the infant brain to acceleration-

⁵² Cory, C.Z. & Jones, M.D., *Can Shaking Alone Cause Fatal Brain Injury? — A Biomechanical Assessment of the Dubaime Shaken Baby Syndrome Model*, 43 Med. Sci. L. 317, 327-328 (2003).

⁵³ Nicole G. Ibrahim, et al., *Physiological and pathological responses to head rotations in toddler piglets*, 27 J. Neurotrauma 1021, 1022 (2010).

⁵⁴ Testimony of Dr. Ronald Uscinski at 72, *State v. Listro* (Conn, March 12, 2010).

⁵⁵ Ann-Christine, et al., *Maturation-dependent response of the piglet brain to scaled cortical impact*, 93 J. Neurosurgery 455-462 (2000).

⁵⁶ Thibault, K.L., & Margulies, S.S. *Age-dependent material properties of the porcine cerebrum: effect on pediatric inertial head injury criteria*, 31 J. Biomechanics 1119 (1998); Nicole G. Ibrahim, et al., *Physiological and pathological responses to head rotations in toddler piglets*, 27 J. Neurotrauma 1021, 1022 (2010).

⁵⁷ Nicole G. Ibrahim, et al., *Physiological and pathological responses to head rotations in toddler piglets*, 27 J. Neurotrauma 1021, 1022 (2010).

⁵⁸ *Id.*

⁵⁹ *Id.*

deceleration movements, compared with the toddler.”⁵⁹

Traditional biomechanical scaling between infants and older children or adults fails to account for differences in myelination and tissue development, and therefore may not reflect the infant brain’s increased susceptibility to rotational injury.⁶⁰ Myelin is a protective coating that develops around the brain and spinal cord’s axons. Although an infant brain is much less myelinated than an adult brain,⁶¹ a defense expert may not agree that the lack of myelination renders a human infant more susceptible to injury. The following exchange also took place with Dr. Uscinski in *State v. Listro*:⁶²

Q: Is that what makes infant heads that much more susceptible to injury? Due to the lack of myelination?

A: No more or less. No, that’s got nothing to do with this injury business. It’s mass, again. Not myelin. Mass.

Despite Dr. Uscinski’s testimony, several piglet studies demonstrate that an infant piglet’s brain, which is similarly unmyelinated, is more vulnerable to rotational acceleration than an adult or toddler pig’s brain.⁶³ In addition to the Ibrahim and Margulies study discussed previously, Raghupathi and Margulies in 2002 found that in comparison to adult pigs, infant piglets exposed to rotational forces suffered greater injury per unit area.⁶⁴ An infant’s brain, due to structural immaturity and chemical differences including the lack of myelin, may be more susceptible to acceleration-deceleration injuries than estimated by Duhaime.

The Duhaime models’ brains were tightly packed with cotton and dissimilar to an infant’s brain structure. While the Duhaime model had a rigid plastic skull, a human infant has a soft skull that could render it at increased risk for injury. In conclusion, infant brains may be more susceptible to injury due to anatomical and chemical differences from toddler and adult brains.

⁶⁰ *Id.*

⁶¹ Mary Case, *Abusive Head Injuries in Infants and Young Children*, 9 St. Louis U. Med. Center, Forensic Pathology, 83-87 (2007).

⁶² *Id.* at 116.

⁶³ Nicole G. Ibrahim, et al., *Physiological and pathological responses to head rotations in toddler piglets*, 27 J. Neurotrauma 1021, 1021 (2010); Thibault, K.L., & Margulies, S.S. *Age-dependent material properties of the porcine cerebrum: effect on pediatric inertial head injury criteria*, 31 J. Biomechanics 1119 (1998).

⁶⁴ Ramesh Raghupathi & Susana S. Margulies, *Traumatic Axonal Injury Following Closed Head Injury in the Neonatal Pig*, 19 J. Neurotrauma 843, 851 (2002).

An adult monkey's head is anatomically distinct from the immature human infant skull and brain, and "extrapolating from the forces required to cause (or not cause) certain injuries in animals to a human infant may not be possible."⁶⁵

Accurate Science: *Duhaime's models were only shaken in a single direction, which does not account for the increased acceleration found by shaking in various directions.*

In addition to multiple shaking cycles, abusive head trauma incidents involve infants being shaken in multiple directions. In Duhaime's study, the models were consistently shaken in the anteroposterior (A-P) direction, and the measurement apparatus only captured accelerations on the A-P plane.⁶⁶ Duhaime's study, therefore, did not account for the fact that shaking in different directions causes varying degrees of acceleration. For instance, sideways movement generates greater rotational forces and creates greater shearing injury to the brain than straight front-to-back shaking.⁶⁷ Cory and Jones accounted for the possibility of infants being shaken in different directions by shaking the models in two types of scenarios. In the first scenario, volunteers held the model under the arms and shook it repeatedly in the anteroposterior direction. In the second scenario, designed to be the "worst case scenario," volunteers shook the model in a "gravity-assisted" shake pattern, "which had been shown to produce the greatest level of acceleration."⁶⁸ This pattern entailed the volunteer holding the model high above one shoulder and then accelerating the model downward below the waist repeatedly.⁶⁹ The results were that the models shaken in the gravity-assisted position achieved considerably higher levels of acceleration than those shaken in anteroposterior direction.

Thus, Cory and Jones concluded from their study that "at this present stage . . . it cannot be categorically stated, from the Duhaime study, that 'pure shaking' cannot cause fatal head injuries in an infant" and that "sufficient doubt in the reliability of the Duhaime biomechanical study [warrants] the exclusion of such testimony in cases of suspected shaken baby syndrome."⁷⁰

⁶⁵ Henry F. Krous & Roger W. Byard, *Controversies in Pediatric Forensic Pathology*, Forensic Sci. Med. Pathology 9, 13 (2005).

⁶⁶ Ann-Christine Duhaime, et al., *The Shaken Baby Syndrome: A Clinical, Pathological, and Biomechanical Study*, 66 J. Neurosurgery 409, 412 (1987).

⁶⁷ Thomas A. Gennarelli & Lawrence E. Thibault, *Biomechanics of Acute Subdural Hematoma*, 22 J. Trauma 680 (1982); Mary E. Case, *Inflicted Traumatic Brain Injury in Infants and Young Children*, 18 Brain Pathology 571 (2008); Cory, C.Z. & Jones, M.D., *Can Shaking Alone Cause Fatal Brain Injury? — A Biomechanical Assessment of the Duhaime Shaken Baby Syndrome Model*, 43 Med. Sci. L. 317 (2003).

⁶⁸ Cory, C.Z. & Jones, M.D., *Can Shaking Alone Cause Fatal Brain Injury? — A Biomechanical Assessment of the Duhaime Shaken Baby Syndrome Model*, 43 Med. Sci. L. 323 (2003).

⁶⁹ *Id.* at 323.

⁷⁰ *Id.* at 332.

Defense Claim:

**It is impossible to cause brain injuries
though shaking absent a concomitant neck injury.**

Defense experts will often testify that violent shaking would injure an infant's neck prior to injuring the brain. Dr. Patrick Barnes testified in 2008 that "[a]ll the recent literature tells us that if shaking only is going to produce this type of brain injury we'd probably have to have neck injury, spine injury or spinal cord injury with it because that's the weakest part of the head and neck."⁷¹ However, many children who are violently shaken do suffer brain injuries without an accompanying neck injury.⁷² In comparison to adults, children have very pliable necks, weak neck muscles, and relatively large heads in comparison to their bodies.⁷³ In fact, it is rare for children to suffer soft tissue or bone injuries to the neck through shaking.⁷⁴ Shaking may produce subtle cervical spinal cord injuries that can be detected during an autopsy, but these injuries are likely to be undetected in children who survive shaking incidents.⁷⁵

Accurate Science: *The defense relies upon a biomechanical study by Faris Bandak that has been heavily criticized in the medical community.*⁷⁶

It is important for the prosecutor to establish that "all the recent literature" includes only the Bandak study, at which point the prosecutor can cross-examine the witness with criticisms of Bandak's study. Despite his "all of the recent literature" testimony during the 2008 trial, Dr. Barnes only named the Bandak study as supporting his testimony about neck injuries.⁷⁷ Barnes did not answer a subsequent question about having omitted reference to Bandak in his pretrial written report.⁷⁸ In a 2007 article Barnes co-authored, he cited two sources for the proposition

⁷¹ Direct Examination of Dr. Patrick Barnes at 43, *State v. Maze*, No. 2002-D-2361(Tenn. Crim. Ct. June 9, 2008).

⁷² Laura K. Brennan et al., *Neck injuries in young pediatric homicide victims*: Clinical article, 3 J. Neurosurgery Pediatrics 232-239 (2009).

⁷³ Lori Frasier, MD et al., *Abusive Head Trauma in Infants and Children: A Medical, Legal, and Forensic Reference*, 167 (G.W. Medical Publishing 2006).

⁷⁴ *Id.* at 167.

⁷⁵ James R. Gill et al., *Fatal head injury in children younger than 2 years in New York City and an Overview of the Shaken Baby Syndrome*, 133 Archives of Pathology & Laboratory Med. 619, 625 (2009).

⁷⁶ Bandak, F.A., *Shaken baby syndrome: A biomechanics analysis of injury mechanisms*, 151 Forensic Sci. Int. 71 (2005).

⁷⁷ Cross Examination of Dr. Patrick Barnes at 72, *State v. Maze*, No. 2002-D-2361(Tenn. Crim. Ct. June 9, 2008).

⁷⁸ *Id.*

that a neck injury would result from shaking before brain injuries; both articles he cited were authored by Bandak.⁷⁹ Dr. Ronald Uscinski presented at a hospital grand rounds in Maryland on April 15, 2010, and discussed the Bandak study, which he also featured in his PowerPoint presentation. However, when he testified the month prior in Connecticut, Dr. Uscinski omitted all mention of Bandak, despite extensively testifying regarding neck injuries.⁸⁰

It seems plausible that defense witnesses prefer not to be cross-examined on the Bandak study's limitations. In 2006, Margulies, Prange, et al., responded to Bandak's study with a letter to the editor stating, "[w]e are gravely concerned that the conclusions reached by Bandak may be invalid due to apparent numerical errors in his estimation of forces. . . More specifically, we have repeated his calculations and we find values of neck forces that are actually more than 10x *lower* than those presented in Bandak's Table 3."⁸¹ "Based upon his flawed calculations, Bandak erroneously concluded that the neck forces in even the least severe shaking event far exceed the published injury tolerance of the infant neck. However, when accurately calculated, the range of neck forces is considerably lower, and includes values that are far below the threshold for injury. In light of the numerical errors in Bandak's neck force estimations, we question the resolute tenor of Bandak's conclusions that neck injuries would occur in all shaking events. Rather, we propose that a more appropriate conclusion is that the possibility exists for neck injury to occur during a severe shaking event without impact."⁸²

In a *Quarterly Update* review,⁸³ pediatrician Betty Spivack criticized the sloppy methodology of Bandak's study. Bandak relied on only a single study involving human subjects. That 1874 study involved suspending weights from the necks of stillborns to determine at what weight the necks would break and decapitation occur.⁸⁴ Spivack noted that while rapid shaking incidents involve tensile loads experienced over short time intervals, Bandak relied upon four studies in which tensile loads were experienced over long intervals. Dr. Spivack concluded "[i]t is inappropriate to use thresholds derived from one sort of loading condition to infer injury under

⁷⁹ Patrick D. Barnes & Michael Krasnokutsky, *Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics*, 18 Topics Magnetic Resonance Imaging 53, 55 (2007).

⁸⁰ Testimony of Dr. Ronald Uscinski at 15, 16, 74-79, 111, *State v. Listro*, No. TTD-CR08-0092447-T (Conn. Super. Ct., March 12, 2010).

⁸¹ Susan Margulies, Michael Prange & Barry Myers, *Letter to the Editor — Shaken baby syndrome: A flawed biomechanical analysis*, 164 Forensic Science 278 (2006).

⁸² Susan Margulies, Michael Prange & Barry Myers, *Letter to the Editor — Shaken baby syndrome: A flawed biomechanical analysis*, 164 Forensic Science 278, 279 (2006).

⁸³ XIII *The Quarterly Update* 1, 25 (Jan. 2006).

⁸⁴ XIII *The Quarterly Update* 1, 25 (Jan. 2006) citing Duncan, J.M., *Laboratory Note: On the Tensile Strength of the Adult Fetus*, 2 Brit. Med. J. 763-764 (1874).

very different conditions.” Dr. Spivack further describes the “extremely sloppy manner in which the medical literature is cited,” and concludes “the lack of attention to important details pertinent to both engineering and medical literature engenders no degree of confidence in the conclusions expressed by Dr. Bandak.”⁸⁵

Accurate Science: *Shaking rarely results in soft tissue and bone injuries to the infant’s neck, and cervical nerve injuries are often present but difficult to detect.*

The most recent and comprehensive article on neck injuries in AHT fatalities shows that neck soft tissue and bone injuries are rare in AHT fatalities. In a study by Brennan, neck injuries were found at autopsy in only nine out of 41 children fatally injured in abusive head trauma cases.⁸⁶ Among those nine victims, six had muscle injuries, three had ligamentous injuries, and another three had other soft-tissue injuries to the neck. Cervical-spinal cord and nerve root injuries were found in 29 out of the 41 fatalities, but these injuries cannot be detected on cat scan (CT) or magnetic imaging (MRI) and require pathologic dissection.⁸⁷ Among children who survive violent shaking, subtle damage to the nerve roots of the neck and spine may not be detected.

In 2011, Matshes found cervical nerve root damage injuries in all 12 children autopsied whose deaths involved either accidental or inflicted whiplash injuries to the neck, but in only one of 23 children whose deaths did not involve whiplash neck injuries.⁸⁸ Matshes utilized a more thorough autopsy procedure, and looked for nerve root injuries that are not visible through standard spinal chord and neck autopsies. Matshes concluded that “up until now, neck injuries have not been seen, not because they were not present, but because the appropriate anatomical structures were not dissected.”⁸⁹

The defense expert may argue that if there were no neck findings, then the victims studied were not in fact injured from shaking. The following exchange involving Dr. Uscinski took place in *State v. Listro*:⁹⁰

⁸⁵ XIII *The Quarterly Update* 1, 25 (Jan. 2006).

⁸⁶ *Id.* at 235.

⁸⁷ *Id.* at 237.

⁸⁸ Matshes, Evan, et al. *Shaken Infants Die of Neck Trauma, Not of Brain Trauma*. Academic Forensic Pathology, Vol 1 (1), 82-91, (July 2011).

⁸⁹ *Id.*

⁹⁰ Testimony of Dr. Ronald Uscinski at 77, *State v. Listro*, No. TTD-CR08-0092447-T (Conn. Super. Ct.. March 12, 2010).

Q: Are you aware of research out there that have found that there is no neck injury in the cases where a child's been fatally injured by head trauma?

A: Yes. That's the point . . . I can't think of one particular study that shows clinically that a baby has had a neck injury.

To counter such an argument, the prosecutor can point out that studies such as Brennan's examine confirmed fatal victims of AHT, and that these victims did not have neck injuries. In instances where the child survives, the prosecutor can cite the studies by Brennan and Matshes and question the defense witness on whether it is possible to detect subtle injury to the cervical spine and nerve root without performing autopsies.

In conclusion, whiplash injuries to the neck, if present, are often subtle, may sometimes only be detected through meticulous dissection of the anterior and posterior neck, and are not necessary for a diagnosis of abusive head trauma.⁹¹

Defense claim: Short falls commonly produce the types of fatal cranial injuries seen in AHT cases.

Accurate Science: *Contact head injuries from falls produce brain injuries that differ from the types of injuries caused by shaking.*

Serious injuries rarely result from short falls, but when they do, they are usually indicated by focal injuries such as epidural hematomas (EDH) that are distinct from the global, diffuse injury typical of fatal abusive head trauma cases.⁹² A much more common serious or fatal head injury in young children is caused by events that produce rotational movement of the brain about its center of gravity.⁹³ In contrast, the impact from a fall causes the brain to move in a linear direction, which causes less damage outside of the focal contact area than when the brain is sub-

⁹¹ James R. Gill et al., *Fatal head injury in children younger than 2 years in New York City and an Overview of the Shaken Baby Syndrome*, 133 Archives Pathology and Laboratory Med. 619, 625 (2009).

⁹² Stocker, Thomas, and Dehner, Louis, *Pediatric Pathology* 268 (2nd ed. 2002).

⁹³ *Id.*

⁹⁴ Mary E. Case, *Inflicted Traumatic Brain Injury in Infants and Young Children*, 18 Brain Pathology 571-582 (2008).

ject to rotational forces.⁹⁴

Accurate Science: *Short falls are frequently provided as false histories when in fact child abuse has occurred.*

Rather than disclose abuse, the abuser may claim that the child's injuries resulted from a short fall. Medical personnel suspect abuse when the child's injuries are inconsistent with the history provided by the caregiver. In 1991, Dr. David Chadwick examined 283 emergency room cases in which parents reported children had suffered falls.⁹⁵ Seven fatalities were recorded among the 100 children reported to have fallen less than four feet. Chadwick's study found only one death resulted from the 183 falls between five to 45 feet. If the caregiver's histories were accepted as accurate, the implication would be that falls from under four feet were eight times as lethal as falls from five to 45 feet. All seven children who died after alleged short falls had fresh subdural bleeding and cerebral edema, and five had other injuries consistent with child abuse (healing fractures, bruises, genital injuries, etc.). Chadwick concluded, "[f]alls of less than four feet are often reported in association with children's head injuries that prove to be fatal, but such histories are inaccurate in all or most such cases."⁹⁶

A study comparing falls corroborated by a second witness with falls witnessed only by the caregiver further illustrates that a false history should be suspected when severe injuries are attributed to a short fall.⁹⁷ In corroborated falls under ten feet, the study by Williams found no life-threatening injuries amongst 106 infants and children. The only death resulted from a 70-foot fall. In uncorroborated falls, severe injuries were common and two deaths were found amongst children alleged to have fallen less than five feet, leading Williams to "suspect that many if not all of these injuries attributed to falls of low height represent child abuse."⁹⁸

The Williams study is consistent with an earlier study by Helfer that found no serious in-

⁹⁵ David L. Chadwick, et al., *Deaths from Falls in Children: How Far Is Fatal*, 31 J. Trauma 1350, 1353 (1991).

⁹⁶ *Id.* at 1355.

⁹⁷ R.A. Williams, *Injuries in Infants and Small Children Resulting from Witnessed and Corroborated Free Falls*, 31 J. Trauma 1350, 1351 (1991).

⁹⁸ *Id.* at 1351.

⁹⁹ Helfer RE, Slovis TL, Black M. *Injuries Resulting When Small Children Fall Out of Bed*. Pediatrics 60: 533-535 (1977).

juries among 85 children who had fallen off of a bed, crib, or examination table.⁹⁹ In 57 incidents the children did not suffer an injury, in 17 incidents children had small cuts, in 20 incidents children had a bump or bruise, and one child had a skull fracture with no apparent sequelae.¹⁰⁰ In a recent study in England, 11 newborns suffered objectively-witnessed falls onto vinyl floors (with underlying concrete) in a hospital.¹⁰¹ Only one child who had fallen over three feet had some neurologic symptoms and a cerebral contusion. None of the children had SDH or significantly poor outcomes.¹⁰² While this is only a case series of 11, it corroborates what Williams found in the 1990s and HeIfel found in the 1970s.

Defense witness Dr. John Plunkett frequently testifies that a short fall may result in catastrophic brain injury. In 2007, Plunkett testified, “[t]he seven week old skull is so easily deformed that with impact, any impact, there’s going to be inbending of the skull. That inbending, if it is high enough, will cause distortion and movement of the underlying brain . . . [t]hat mass movement may cause stretching of bridging veins.”¹⁰³

Plunkett bases his testimony in part upon his own 2001 study of 75,000 playground falls, 18 of which resulted in death.¹⁰⁴ However, Plunkett’s study did not include any infants, and does not prove that a short fall can kill an infant. Studies suggest that the infant brain, while more susceptible to rotational injury,¹⁰⁵ is more resistant to impact injuries.¹⁰⁶ In Plunkett’s study, nine of the 18 children who died were above the age of five, and none of the children was younger than 12 months of age. Additionally, some of the falls were accelerated because the children were swinging from playground equipment. Other falls were from seven feet, and therefore not “short falls.”¹⁰⁷ Several children had pre-existing conditions that may have increased their risk of death.¹⁰⁸ Even accepting Plunkett’s conclusions, the rate of death was less than 0.02 percent.

Defense witnesses will also cite a single case study documented by Denton and Mileusnic.¹⁰⁹ A grandmother claimed that a nine-month-old child fell off of a bed, acted nor-

¹⁰⁰ *Id.*

¹⁰¹ Ruddick C et al. *Head Trauma Outcomes of Verifiable Falls in Newborn Babies* Arch Dis Child Fetal Neonatal Ed Mar 95 (2): F144-5 (2010).

¹⁰² *Id.*

¹⁰³ Excerpt of Testimony of Dr. John Plunkett at 21, *State. v. Scott* (Arizona 2007) (Contact NCPA for this transcript).

¹⁰⁴ John Plunkett, *Fatal Pediatric Head Injuries Caused by Short-Distance Falls*, 22 Am. J. Forensic Med. Pathology 1, 1 (2001).

¹⁰⁵ N. Ibrahim, J. Ralston, C. Smith, S. Margulies, *Physiological and Pathological Responses to Head Rotations in Toddler Piglets*, 27 J. Neurotrauma 1021-1035 (2010).

¹⁰⁶ Ann-Christian Duhaime et al., *Maturation-Dependent Response of the Piglet Brain to Scaled Cortical Impact*, 93 J. Neurosurgery 455-462 (2000).

¹⁰⁷ Robert M. Reece, Letter to the Editor, *The evidence base for shaken baby syndrome: Response to editorial from 106 doctors*, 328 Brit. Med. J. 1316-1317 (May 2004).

¹⁰⁸ *Id.*

¹⁰⁹ Denton, Scott and Mileusnic, Darinka, *Delayed Sudden Death in an Infant Following an Accidental Fall*, 24 Am. J. Forensic Med. Pathology 371 (2003).

mally for the next several days, and was then found dead. Among other injuries, the child's autopsy showed a posterior parietal skull fracture and a subdural hematoma. This study is relied upon by defense experts to support both the short fall and lucid interval defense. However, the Denton study is based on a single case report of an unwitnessed fall. Although there was no indication of child abuse or dishonesty, there is cause to be skeptical given the inconsistency between the given history and the child's injuries.¹¹⁰ In addition, the injuries documented in this case study—including a large focal subdural bleed in the posterior of the skull—are dissimilar to the diffuse global injuries typical in AHT cases.

Accurate Science: *The risk of a short fall causing fatal injuries in infants is less than one in a million.*

In 2008, Dr. Chadwick conducted a meta-analysis explicitly to ascertain the mortality rate of children under the age of five who fell short distances (under three feet).¹¹¹ Chadwick found that the risk of a fatal injury was 0.48 children per million in cases of reported short falls. This study included only reported falls, not minor falls that parents did not report, and therefore the rate of death following reported and unreported falls is obviously even lower than 0.48 per million. Studies in a variety of settings confirm that short falls do not result in death or serious injury to children. In a study of 207 children under five years old who fell from hospital beds, Lyons and Oates found no resulting brain injuries.¹¹² In large daycare centers, where a child's fall is likely to be witnessed, there is not a single published, peer-reviewed, medical account of a child dying from a fall.¹¹³ A review of the literature by Dr. Rieber concludes "[m]ajor injuries nearly always result from major impacts and serious falls."¹¹⁴

¹¹⁰ Krous, Henry F. and Byard, Roger W. *Controversies in Pediatric Forensic Pathology*, 1 Forensic Sci. Med. Pathology 9, 13 (2005).

¹¹¹ Chadwick, David L., et al., *Annual Risk of Death Resulting from Short Falls Among Young Children: Less Than 1 in 1 Million*, 121 Pediatrics 1213-1224 (2008).

¹¹² Lyons, T.J. and Oates, R.K., *Falling Out of Bed: A Relatively Benign Occurrence*, 92 Pediatrics 125-127 (1993).

¹¹³ Chadwick, David L., et al., *Annual Risk of Death Resulting from Short Falls Among Young Children: Less Than 1 in 1 Million*, 121 Pediatrics 1213, 1216 (2008).

¹¹⁴ Reiber, Gregory D. *Fatal Falls in Childhood: How Far Must Children Fall to Sustain Fatal Head Injury? Report of Cases and Review of the Literature*, 14 Am. J. Forensic Med. Path. 201, 207 (1993).

Defense Claim:

A hypoxic rather than traumatic event might cause subdural hemorrhage.¹¹⁵

The defense witness will testify that a lack of oxygen, known as “hypoxia,” can cause a subdural hematoma. The defense witness may claim that “ischemia” (damage caused by a lack of blood flow), or “anoxia” (the absence of oxygen) can cause subdural hematomas and will likely cite research by Dr. Jennian Geddes. An example of such testimony is below:

Q: . . . [T]here were advancements in the literature about ischemic injury and hypoxia; is that what you’re saying?

A: There’s been advancements in the literature about how we use the term “diffuse axonal injury,” and I think most of us are a little bit more careful to make a distinction between whether it’s due to low oxygen or whether it’s due to impact.¹¹⁶

Accurate Science: *Geddes’s research is highly experimental, and does not demonstrate that hypoxia causes subdural hematomas.*

Geddes’s highly experimental, and conventional theory suggests that hypoxia is in fact a result rather than a cause of brain and spinal injuries in AHT cases. Dr. Geddes herself somewhat recanted her theory during the 2005 legal proceedings in England involving challenges to “shaken baby syndrome.”

¹¹⁵ Geddes, J.F. et al, *Dural haemorrhage in non-traumatic infant deaths: does it explain the bleeding in ‘shaken baby syndrome’?*, 29 *Neuropathology and Applied Neurobiology* 14, 14 (2003).

¹¹⁶ Excerpt of Testimony of Janice Ophoven at 23, *State v. Helms*, No. RIF-102020 (March 4, Riverside California 2004). (Contact NCPCA for this transcript).

A: I think we might not have the theory quite right. I think possibly the emphasis on hypoxia—no, I think possibly we are looking more at raised pressure being the critical event.

Q: Dr Geddes, cases up and down the country are taking place where Geddes III is cited by the defense time and time again as the reason why the established theory is wrong.

A: That I am very sorry about. It is not fact; it is hypothesis but, as I have already said, so is the traditional explanation. ... I would be very unhappy to think that cases were being thrown out on the basis that my theory was fact. We asked the editor if we could have “Hypothesis Paper” put at the top and he did not, but we do use the word “hypothesis” throughout.”¹¹⁷

Accurate Science: *Courts and subsequent research have rejected hypoxia as a cause of subdural hematoma:*

Although hypoxia can cause cerebral edema, survivors of inflicted head trauma do not typically have drastic brain edema or severely elevated intracranial pressure.¹¹⁸ A number of British courts have found that testimony that hypoxia causes subdural hematomas to be unsubstantiated.¹¹⁹ Several studies have examined the relationship between hypoxia and SDH and the results directly refute Geddes’ “Unified Theory.”¹²⁰ A 2010 study included infants and children under the age of four dying in the emergency department or admitted to the pediatric intensive case unit (PICU) after atraumatic cardiorespiratory arrest.¹²¹ Of those children who experienced a cardiorespiratory arrest from a non-traumatic cause and met inclusion criteria, 33 presented and died in the ED and 17 were admitted to the PICU. These children had a post-mortem examination, brain imaging or both. None of these infants had a significant SDH. One child had a small clot adherent to the dura found on post-mortem and two had microscopic intradural hemorrhage, but it is unclear in each case whether this was an artifact of the autopsy,

¹¹⁷ Transcript, *R. v Harris, Rock, Cherry, and Faulder* (Court of Appeal, Royal Courts of Justice, Strand, London, United Kingdom; June 17, 2005).

¹¹⁸ Minns, Robert A. and Brown, Keith J. *Shaking and Other Non-Accidental Head Injuries in Children*, 161 *Archives of Pediatrics and Adolescent Med.* 108, 258 (2007).

¹¹⁹ *Regina v Harris*_2005; *A Local Authority v S* [2009] EWHC 2115 (Fam).

¹²⁰ Byard, Roger W. et al., *Lack of Evidence for a Casual Relationship Between Hypoxic-Ischemic Encephalopathy and Subdural Hemorrhage in Fetal Life, Infancy, and Early Childhood*, 10 *Pediatric and Developmental Pathology*, 348, 349 (2007); Punt, J. et al., *The ‘unified hypothesis’ of Geddes et al is not supported by the data*, 7 *Pediatric Rehabilitation* 173 (2004) (This is a review article).

¹²¹ Hurley et al., *Is there a causal relationship between the hypoxia-ischaemia associated with cardiorespiratory arrest and subdural haematomas? An observational study*. *Br J Radiol Sep*, 83 (993) 736-743 (2010).

as each had otherwise normal brains. The author's concluded "Subdural hematoma arising in infants or young children in the context of catastrophic cardiorespiratory compromise from a non-traumatic cause was not observed."¹²²

Defense Claim:

**A child's previously existing subdural hematoma may "rebleed,"
either spontaneously or as a result of a trivial injury,
and cause severe brain injuries.**

Defense witnesses may testify that a child's acute symptoms were not the result of an immediate injury, but rather a "rebleed" of a chronic subdural hematoma. In such a case, the defense expert may testify that there is radiological evidence of "old" blood mixed with "new" blood, which indicates that an existing subdural hematoma began to bleed again. The defense witness may claim the child's initial subdural hematoma was produced through the birth process, a past traumatic injury that was clinically silent, or a hypoxic event. That witness will then claim that the previous subdural hematoma never properly healed, and began to rebleed. Below are examples of defense witness testimony regarding rebleeds:

- "The original injury may actually have been a trivial injury that wasn't appreciated. But the normal healing mechanism went awry and the subdural didn't go away."¹²³
- "The process of absorption of a subdural hematoma is a dynamic process. It is absorbing and rebleeding at the same time. And if you're absorbing faster than you're re-bleeding you get better. If you're re-bleeding faster than you're absorbing, you get worse."¹²⁴

¹²² *Id.*

¹²³ Transcript of Record, Testimony of Ronald Uscinski at 29, *State v. Listro* No. TTD-CR08-0092447-T (Conn. Super. Ct. March 12, 2010).

¹²⁴ Interview of Dr. Uscinski at 15, *State v. Cayce Brooks* (2000). (Contact NCPA for a copy of this transcript).

-
- Regarding what force would be necessary to cause a rebleed, Dr. Uscinski has testified to the following: “hopping on one foot, coughing, sneezing, straining at having a bowel movement, bouncing a baby up and down on your knee if a baby has a chronic subdural.”¹²⁵

Accurate Science: *There is no evidence that an infant’s subdural hematoma resulting from a traumatic event will spontaneously rebleed as a result of a minor injury.*

According to Duhaime, “[t]here is no evidence that traumatic acute subdural hematoma, particularly that lead to death, occurs in otherwise healthy infants in an occult or subclinical manner.”¹²⁶ A rebleed results in low-pressure venous blood, and therefore should not cause neurological deterioration or other acute clinical features.¹²⁷ In addition, a rebleeding chronic subdural hematoma would be indicated by a gradual onset of symptoms including lethargy and irritability, not the rapid-onset symptoms characteristic of AHT cases.¹²⁸ While a subdural hematoma caused by trauma could rebleed, a second “shaking episode would need to be about as forceful as the first to cause significant damage.”¹²⁹

The defense witness may also reference a study by Hymel to suggest that a traumatic subdural hematoma may rebleed.¹³⁰ There are several important points from the two case studies documented by Hymel. First, both children were over 11 months of age, and their hematomas were unrelated to the birth process. In the first case, the child fell down several concrete stairs and suffered a skull fracture and epidural hematoma. Three months later he hit his head on a windowsill and suffered a rebleed. However, because the second trauma was minor, his symptoms were limited to irritability and a loss of appetite.¹³¹ In the second case study, the 11-month-old child suffered more than a minor injury when he fell out of the hospital bed, and was already symptomatic prior to the fall.¹³² The Hymel article does

¹²⁵ Excerpt of Testimony of Dr. Ronald Uscinski at 21, *State v. Cutro* (South Carolina 1999). (Contact NCPA for a copy of this transcript).

¹²⁶ Ann-Christine Duhaime et al., *Nonaccidental Head Injury in Infants—The Shaken-Baby Syndrome*, 338 New Eng. J. of Med. 1822-29, 1825 (1998).

¹²⁷ Lori Frasier et al., *Abusive Head Trauma in Infants and Children: A Medical, Legal, and Forensic Reference* 61 (G.W. Medical Publishing, Inc. 2006).

¹²⁸ Hymel, Kent P. et al., *Intracranial Hemorrhage and Rebleeding in Suspected Victims of Abusive Head Trauma: Addressing the Forensic Controversies*, 7 Child Maltreatment 329, 340 (2002).

¹²⁹ Alexander, Randell C., *Review of Robert C. Cantu, Second-Impact Syndrome*, 17 Clinical Sports Med. 37-44, (1998); 5 Child Abuse Quarterly Medical Update 11, 12 (1998).

¹³⁰ Hymel, Kent P. et al., *Intracranial Hemorrhage and Rebleeding in Suspected Victims of Abusive Head Trauma: Addressing the Forensic Controversies*, 7 Child Maltreatment 329-348 (2002).

¹³¹ *Id.* at 331-332.

¹³² *Id.* at 342-343.

demonstrate that a subdural hematoma could rebleed in a child, but that if new symptoms are seen, there must have been significant new brain injury.

Accurate Science: *The literature does not support the notion that an infant's chronic subdural hematoma can spontaneously rebleed.*

Defense experts often cite studies showing that some infants are born with subdural hematomas. For instance, the defense may refer to a 2008 study by Rooks in which 46 out of 101 newborns had SDH at birth.¹³³ The defense may also cite studies showing chronic subdural hematomas can rebleed without additional trauma.¹³⁴ The defense expert will often offer this testimony without providing the context of the studies, or will advance further assertions that are not supported by the cited literature. For instance, the defense may cite studies showing chronic subdural hematomas in adults can rebleed, and then without basis extrapolate from these studies that an infant's SDH from birth could similarly rebleed. Dr. Uscinski often relies on a number of Japanese studies from the 1970s in which chronic subdural hematomas in adult patients spontaneously rebled.¹³⁵ In particular, it appears that Uscinski is relying on the work of a Japanese neurosurgeon Haruhide Ito to support his testimony.¹³⁶ However, the Ito studies were conducted with adults, not children, and do not support the proposition that SDH in infants can rebleed and cause brain injury. Prosecutors should challenge the defense witness to name a single study showing that a child's chronic subdural hematoma can rebleed from a trivial injury and cause brain injuries. The following exchange took place between the prosecutor and Dr. Uscinski in a 2009 case:

Q: Could I have the name of the article or the research that you've said there was?

A: I gave you one. Ito. There's another one by Ito in 1978.

¹³³ Rooks et al., *Prevalence and Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants*, 29 Am. J. Neuroradiology, 1082, 1083 (2008).

¹³⁴ Uscinski, Ronald H. Letters to the Editor, *Neurosurgical Forum*, 105 J. Neurosurgical Pediatrics, 333 (2006).

¹³⁵ See e.g., Excerpt of Testimony by Uscinski at 26-27, *State v. Listro*, No TTD-CR08-0092447 (Conn. Super. Ct. March 12, 2010).

¹³⁶ Ito, Komai, and Yamamoto, *Fibrinolytic Enzyme in the Lining Walls of Chronic Subdural Hematoma*-48 J. Neurosurgery (1978); Ito, Yamamoto, Komai, Mizukoshi, *Role of Local Hyperfibrinolysis in the Etiology of Chronic Subdural Hematoma* 45 J. Neurosurgery (1976).

Q: Applying to infants was my question. Please tell me the name of that research article.

A: I just did. It applies to infants and adults.

Q: But there were no infants in the Ito study.

A: That's correct.¹³⁷

Accurate Science: *If the birth process causes subdural bleeding in an asymptomatic newborn, the subdural bleed will quickly resolve without ever becoming symptomatic.*

Subdural bleeds in newborns are small (<3mm), lie primarily in the posterior fossa (the back part of the brain)—in contrast to the diffuse SDH that typically result from rotational trauma—and resolve without rebleeding or becoming symptomatic.¹³⁸ The defense witness may rely on a number of studies, including those by Rooks, Whitby, and Looney, to establish that the birth process can produce subdural hematomas, without mentioning that none of the children in these studies were ever noted to become symptomatic. In Rooks, 46 out of 101 children had SDH at birth. However, 16 children who had an SDH at birth were reimaged at one month, and in 15 cases the SDH had completely resolved.¹³⁹ At three months, all 18 of the children who were reimaged showed complete resolution of the SDH.¹⁴⁰ All 43 patients who submitted to a follow-up at two years demonstrated no gross motor delay.¹⁴¹ Rooks concluded, “hemorrhages seen in asymptomatic term neonates are limited in size and location. SDH after one month of age is unlikely to be birth-related.”¹⁴² In the Whitby study, nine of the 111 newborns who underwent an MRI were discovered to have a subdural hemorrhage. Those nine newborns were re-imaged at four weeks and all hematomas were resolved.¹⁴³ Looney did not perform follow-up imaging to determine the clinical significance of birth-related SDH, but noted “it is likely that small subdural hemorrhages resolve quickly without substantial consequence.”¹⁴⁴ None of the children with birth-related subdural bleeds in the Rooks, Whitby, or Looney studies was ever noted to become symptomatic. Statements by defense witnesses that subdural

¹³⁷ Transcript of Record, Testimony of Dr. Ronald Uscinski at 38, *State v. Hancock* (Wis. Cir. Ct. April 6, 2009) (pm session).

¹³⁸ Rooks, V.J. et al., *Prevalence and Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants*, 29 Am. J. Neuroradiology 1082 (2008).

¹³⁹ *Id.* at 1085.

¹⁴⁰ *Id.*

¹⁴¹ *Id.*

¹⁴² *Id.* at 1088.

¹⁴³ Whitby, E. et al., *Frequency and Natural History of Subdural Hemorrhages in Babies and Relation to Obstetric Factors*, 363 Lancet 846 (2004).

¹⁴⁴ Looney, C. et al., *Intracranial Hemorrhage in Asymptomatic Neonates: Prevalence on MR Images and Relationship to Obstetric and Neonatal Risk Factors*, 242 Radiology 535, 540 (2007).

hematomas resulting from birth persist beyond six to eight weeks—“I personally followed one now for of a year”¹⁴⁵—are not supported by the literature.¹⁴⁶

Accurate Science: *The “rebleed” theory is not accepted by the vast majority of the medical community.*

In a 2005 review of abusive head trauma literature, Krous and Byard wrote “we are unaware of any publications or well-documented examples of the sudden onset of catastrophic (as opposed to gradual) clinical deterioration and sudden death in an infant whose chronic subdural hematoma was caused by inflicted injuries and then complicated by rebleeding.”

After the Louise Woodward trial, 50 physicians signed a letter stating the following: “The hypothesis put forward by the defense that minor trauma caused a ‘rebleed’ of an earlier head injury can best be characterized as inaccurate, contrary to vast clinical experience and unsupported by any published literature . . . [t]he rebleed theory in infants is a courtroom diagnosis, not a medical diagnosis, and the jury properly rejected it.”¹⁴⁷

Defense Claim:
The infant may have suffered
a head injury without manifesting symptoms
until hours or days later—a “lucid interval.”

The defense witness may testify that the child experienced a “lucid interval,” meaning that a child sustained a head injury, was symptomatic or not, improved, then suffered a loss or reduction of consciousness at another point in time remote from the event that started the process. Below are some examples of defense expert testimony regarding lucid intervals.

¹⁴⁵ Testimony of Dr. Ronald Uscinski at 92, *State v. Hancock*, No. 07-CF-2381 (Wis. Cir. Ct. April 6, 2009). (Available through NCPCA).

¹⁴⁶ Krous, Henry F. and Byard, Roger W. *Controversies in Pediatric Forensic Pathology*, 1 Forensic Sci. Med. Pathology 9, 13 (2005).

¹⁴⁷ Amaya M, Bechtel K, Blatl SD, et al. [50 signators], *Shaken baby syndrome and the death of Matthew Eappen*, via internet <http://silcon.com/~ptave/shaken.htm>. (November 11, 1997). (Now available through NCPCA).

-
- “[T]he term that we’re not talking about here is lucid interval People can suffer irreversible injuries to their brain and have a period of time where they are awake, walking and talking and interacting with people until the swelling is so severe or seizures come on.”¹⁴⁸
 - “This particular phenomenon has been explained under a name called malignant cerebral edema, in which a process evolves in the young brain, unbeknownst to anybody . . . some of these cases have come and gone; that is, been away, drifted off into a comatose state, and then surfaced again, kind of a bouncing phenomenon, and then experienced a fatal turn for the worse.”¹⁴⁹

Accurate Science: *There is no evidence of infants having lucid intervals following non-contact head injuries that lead to death.*

Children with acceleration-deceleration brain injuries leading to death or coma are immediately symptomatic and without a lucid interval. A 1997 study of 95 cases of fatal head injury in children revealed only two lucid intervals, and those cases involved epidural, not subdural, hematoma.¹⁵⁰ The children in this study ranged from 99 days to 16 years old. Willman, the study’s author, found that “[e]xcept in cases involving epidural hematomas, the time of injury in a fatal head injury case can be restricted to after the last period of normal consciousness for a child.”¹⁵¹ Willman utilized a relatively small but reliable sample, as 93 of the 95 children were injured in car accidents and their status was assessed both at the scene and a later time by medical professionals. Willman concluded that “[i]f a history purports a lucid interval, . . . that history is likely false and the injury is likely inflicted.”¹⁵²

Perpetrator confessions also contradict the notion of a lucid interval in AHT cases. Star-

¹⁴⁸ Testimony of Janice Ophoven at 58, *California v. Helms* (Riverside, Cal. March 4, 2004). (Available through NCPCA)

¹⁴⁹ Testimony of Jan Leestma at 750, *Colorado v. Fritz*, (2006). (Available through NCPCA).

¹⁵⁰ Willman, Krista et al., *Restricting the Time of Injury in Fatal Inflicted Head Injuries*, 21 Child Abuse and Neglect 929, 934 (1997).

¹⁵¹ *Id.* at 939.

¹⁵² *Id.* at 929.

ling (2004) examined 81 cases of confessed AHT injuries, in which 80 victims had SDH and the other victim had a subarachnoid hemorrhage.¹⁵³ In 57 of the 81 cases, the timing of the injuries could be reliably ascertained.¹⁵⁴ In 52 of those 57 cases, the perpetrators admitted that the symptoms appeared “immediately” after the abuse.¹⁵⁵ In three of the five remaining cases, the children were unobserved immediately after the injuries, but were “severely symptomatic” when next observed.¹⁵⁶ In the other two cases, the caregivers gave inconsistent histories, but in both cases it could be determined that symptoms appeared within 24 hours of the initial injury.¹⁵⁷ This study is consistent with previous confession studies showing the immediate appearance of symptoms in AHT cases.¹⁵⁸

Accurate Science: *Lucid intervals are primarily associated with epidural, not subdural hematomas.*

Lucid intervals are classically associated with epidural hematomas (EDH). EDH is a bleed on the outside of the dura, and is caused almost exclusively by trauma. Unlike a diffuse SDH, which is often a marker of underlying brain trauma, EDH may involve little underlying brain damage outside of the EDH. Epidural hematomas result from contact forces, often falls, and may not trigger an immediate loss of consciousness if there is minimal underlying brain trauma.¹⁵⁹ As the epidural hematoma expands, it may produce a mass effect and result in brain injury and loss of consciousness.¹⁶⁰ The time between the initial injury and the onset of symptoms is the “lucid interval.”

In contrast, a subdural hematoma in an abusive head trauma case is usually not the cause of death, but rather a marker of diffuse underlying brain trauma. Subdural hematomas resulting from acceleration-deceleration injuries are generally thin and rarely space occupying lesions, and therefore do not cause mass effect.¹⁶¹ The difference is that whereas the epidural hematoma is causing the symptoms by putting the pressure on the brain, the subdural hematoma is itself

¹⁵³ Starling, Suzanne. et al., *Analysis of Perpetrator Admissions to Inflicted Traumatic Brain Injury in Children*, 158 Archives of Pediatric and Adolescent Med. 454, 455 (2004).

¹⁵⁴ *Id.* at 456.

¹⁵⁵ *Id.*

¹⁵⁶ *Id.*

¹⁵⁷ *Id.*

¹⁵⁸ See Starling, Suzanne, Holden, J.R. and Jenny, C. *Abusive Head Trauma: The Relationship of Perpetrators to Their Victims*, 95 Pediatrics 259 (1995).

¹⁵⁹ Carlos and Abramowsky et al., *Pediatric Pathology* 269 (J. Thomas Stocker & Louis P. Dehner eds. Lippincott Williams & Wilkins 2nd ed. 2002).

¹⁶⁰ Gill, James et al., *Fatal head Injury in Children Younger Than 2 Years in New York City and an Overview of the Shaken Baby Syndrome*, 133 Archives Pathology and Laboratory Med. 619, 621 (2009).

¹⁶¹ *Id.*

a symptom of underlying brain trauma. The typical SDH that result from rotational forces are small and noncompressive, and therefore do not cause a loss of consciousness.¹⁶² These SDH are “not acutely dangerous, cause few signs or symptoms, and alone do not impair consciousness.”¹⁶³ A study by Greenes and Schutzman revealed that in all 14 infants who presented to an ER with asymptomatic intracranial bleeding, the bleeding resolved without the child ever experiencing symptoms.¹⁶⁴

Defense Claim:

“The best study, of which I’m aware, says that 75 percent will occur within the first 24 hours, and the remaining 25 percent can take up to three or four days.”¹⁶⁵

In the testimony quoted above, defense witness Dr. Peter Stephens is testifying regarding the timeframe for the onset of symptoms after the initial head injury. Stephens’s pretrial report indicated that he was referring to a study by Gilliland. This is an example of how the defense will broaden the definition of “lucid” to include children who are in fact having neurological symptoms—such as vomiting or lethargy—but who are not comatose or nonresponsive.

The Gilliland study actually supports rather than refutes the notion that children who suffer fatal or severe brain injuries are immediately symptomatic.¹⁶⁶ Gilliland’s study was not a measure of the time between the initial injury and the appearance of any symptoms, but rather between the initial injury and the appearance of “severe symptoms.” Gilliland defined “severe symptoms” as “the time when an external event occurred or the caretaker called for medical assistance.”¹⁶⁷ The “symptoms” in the Gilliland study were unresponsiveness, difficulty breathing, or cardiorespiratory collapse.¹⁶⁸

The “reliable” cases in Gilliland’s study were those in which an independent observer —

¹⁶² *Id.*

¹⁶³ *Id.*

¹⁶⁴ Greenes, David and Schutzman, Sara, *Clinical Indicators of Intracranial Injury in Head-Injured Infants*, 104 *Pediatrics* 861 (1999).

¹⁶⁵ Testimony of Dr. Peter Stephens at 47, *United States v. Lloyd*, No. 2009 CF3 26935 (D.C. Super. Ct. August 31, 2010).

¹⁶⁶ Gilliland, M.G.F., *Interval Duration Between Injury and Severe Symptoms in Nonaccidental Head Trauma in Infants and Young Children*, 43 *J. Forensic Sci.* 723 (1998).

¹⁶⁷ *Id.* at 723.

¹⁶⁸ *Id.*

someone other than the caretaker — witnessed the child’s injury. In every such case Gilliland documented, children who sustained a head injury were immediately symptomatic. The cases in which there was a reported “lucid interval” were cases in which the caretaker was the only witness to the event.¹⁶⁹ As Gilliland noted, “in all cases in which the children were seen by an independent observer after injury, they were described as not normal during the interval.”¹⁷⁰ Nothing in Gilliland’s study suggests that children sustain injuries, act normally, and then rapidly deteriorate later. The unreliable cases in Gilliland’s study were those in which the caregiver was the only witness to the injury. Some of the unreliable cases were likely the result of abuse, but the caregiver was the only witness and falsely ascribed the injury to an event occurring hours or days prior to the appearance of “severe symptoms.” In the Gilliland study, relying on these false histories artificially extended the window of time between the reported injuries and appearance of symptoms. In *Lloyd*, excerpted above, the defense witness utilized the broad definition of “lucid” and ignored the effect of the unreliable cases to make claims that are not supported by Gilliland’s study.¹⁷¹

The defense may also rely on John Plunkett’s 2001 study of children who were injured on playground equipment, even though that study did not involve infants.¹⁷² Plunkett’s study examined the reports of over 75,000 child falls from playground equipment. Out of 114 total deaths, only 18 fatalities were the result of head injury caused by short falls.¹⁷³ Plunkett determined that 12 of the 18 children who experienced a fatal fall had a lucid interval. However, no children in the study were under a year of age, and all of the lucid intervals documented in children under age four were less than 15 minutes.¹⁷⁴ Traditional mass scaling fails to account for chemical and physiological differences that cause infant and toddler brains to react differently to injury.¹⁷⁵ Therefore it is inappropriate to apply Plunkett’s data to infants, given that none of the fatal falls involved infants. The infant brain, while more susceptible to rotational injuries,¹⁷⁶ is more resistant to impact injuries.¹⁷⁷

Plunkett never defines “lucid,” “interval,” or “lucid interval,” and does not indicate

¹⁶⁹ *Id.* at 724.

¹⁷⁰ *Id.*

¹⁷¹ Testimony of Dr. Peter Stephens at 47, *United States v. Lloyd*, No. 2009 CF3 26935 (D.C. Super. Ct. August 31, 2010).

¹⁷² Plunkett, John, *Fatal Pediatric Head Injuries Caused by Short-Distance Falls*, 22 Am. J. Forensic Med. Pathology 1 (2001) (All fatalities studied were children over 12 months of age).

¹⁷³ *Id.* at 2.

¹⁷⁴ *Id.* at 3.

¹⁷⁵ Ibrahim, Nicole et al., *Physiological and Pathological Responses to Head Rotations in Toddler Piglets*, 27 J. Neurotrauma 1021, 1033 (2010).

¹⁷⁶ Case, Mary E., *Inflicted Traumatic Brain Injury in Infants and Young Children* 18 Brain Pathology 571-582 (2008).

¹⁷⁷ Duhaime, Anne-Christian, et al., *Maturation-Dependent Response of the Piglet Brain to Scaled Cortical Impact*, 93 J. Neurosurgery 455 (2000).

whether children who were conscious were otherwise symptomatic.¹⁷⁸ Of the 18 fatalities in Plunkett's study, ten were between the ages of one to five years old.¹⁷⁹ Five of the ten children between the ages of one and five who died suffered unwitnessed injuries, including all four children under the age of 23 months.¹⁸⁰ Only five of those ten children between the ages of one and five had autopsies.¹⁸¹ Of the 18 fatalities, six were suffered by children playing on swings.¹⁸² Five of those six children were immediately unconscious, and the other child was described as groggy.¹⁸³ Falls from swings would expose the child to rotational forces in addition to any translational forces, and would be more likely to result in diffuse brain injury. Thus the fact that all six children who died after falling from swings were without a lucid interval supports that children who suffer diffuse brain injury from rotational forces do not have a lucid interval.

The defense may also cite a study by Arbogast that establishes that in very rare instances a child may suffer an ultimately fatal injury without immediately becoming unresponsive or comatose.¹⁸⁴ The Arbogast study assessed children using the Glasgow Coma Scale (GCS). The GCS is a measure of responsiveness used to ascertain alertness following an injury. Two percent of the children in the Arbogast study presented with a Glasgow score above 12, which the authors set as the threshold for being "lucid."¹⁸⁵ The authors caution that the GCS is based on motor and verbal skills that cannot be assessed accurately in children less than 36 months of age, precisely the population at issue in AHT cases. Moreover, the GCS does not assess clinical signs of head trauma, such as vomiting, irritability, or subtle signs of alertness, and the authors caution that a score above 12 "does not imply that these children were completely asymptomatic."¹⁸⁶ The Arbogast study does not suggest that infants can suffer an injury, be completely free of symptoms, and then rapidly and drastically decompensate. Instead, the Arbogast study establishes that in rare instances children who suffer fatal head injuries may not be immediately comatose.

In sum, "the vast majority and the most reliable evidence to date indicates that infants who sustain lethal inflicted as opposed to accidental head trauma experience neurological de-

¹⁷⁸ Plunkett, John, *Fatal Pediatric Head Injuries Caused by Short-Distance Falls*, 22 Am. J. Forensic Med. Pathology 1 (2001).

¹⁷⁹ Byard, Roger W., *Sudden Death in Infancy, Childhood and Adolescence*, 85 (Cambridge University Press, 2nd edition, 2004).

¹⁸⁰ *Id.* at 86.

¹⁸¹ *Id.*

¹⁸² Spivack, Betty, *Fatal Pediatric Head Injuries Caused by Short-Distance Falls* (Letter to the Editor), 22 Am. J. Forensic Med. Pathology 332 (2001).

¹⁸³ *Id.*

¹⁸⁴ Arbogast, Kristy, Marguiles, Susan, Christian, Cindy, *Initial Neurologic Presentation in Young Children Sustaining Inflicted and Unintentional Fatal Head Injuries*, 116 Pediatrics 180 (2005).

¹⁸⁵ *Id.* at 181.

¹⁸⁶ *Id.* at 183.

terioration and loss of consciousness very rapidly, if not immediately following, craniocerebral trauma.”¹⁸⁷ This position is supported by an overwhelming consensus of the medical community. The American Academy of Pediatrics’ Committee on Child Abuse and Neglect has noted that the “clinical signs of shaken baby syndrome are immediate and identifiable as problematic, even to parents who are not medically knowledgeable.”¹⁸⁸

Defense Claim:

Retinal hemorrhages are nonspecific injuries and do not indicate child abuse.

Defense witnesses may minimize the overwhelming correlation between child abuse and retinal hemorrhages and suggest an alternate cause for RH, such as in the testimony below:

“There’s really only two mechanisms for retinal hemorrhage, one is an increase in intracranial pressure above venous pressure . . . the other distinct mechanism is, is structural occlusion of the veins. A blood clot in the veins, for example, a condition called cortical venous thrombus, will cause retinal hemorrhage.”¹⁸⁹

Accurate Science: *Retinal hemorrhages, particularly when multilayered, bilateral, and covering the whole retina to its edges, are overwhelmingly associated with abusive head injury characterized by repetitive acceleration-deceleration injury with or without blunt head trauma.*

A presentation at the 2010 American Association for Pediatric Ophthalmology and Strabismus Annual Meeting analyzed the results of 62 studies of pediatric retinal hemorrhages published since 1965.¹⁹⁰ All of the children studied were younger than 11 years of age, had

¹⁸⁷ Krous, H., Byard, R., *Controversies in Pediatric Forensic Pathology*, 1 Forensic Sci. Med. and Pathology 9-18 (2005).

¹⁸⁸ American Academy of Pediatrics: Committee on Child Abuse and Neglect, *Shaken Baby Syndrome: Rotational Cranial Injuries—Technical Report*, 108 Pediatrics 206, 207 (2001).

¹⁸⁹ Testimony of John Plunkett at 39-40, *Arizona v. Fred Scott* (2007). (Available through NCPCA).

undergone a detailed examination by an ophthalmologist, had retinal hemorrhages due to confirmed AHT (n = 363) or confirmed non-AHT (n = 465), and were without any retinal comorbidity. Seventy-eight percent of patients with confirmed AHT had retinal hemorrhages, in comparison to just 5.3 percent of the non-AHT patients. In the AHT patients with retinal hemorrhages, 83 percent of the hemorrhages were bilateral, while just eight percent of non-AHT patients with retinal hemorrhages had bilateral hemorrhages. For AHT patients, 63 percent of the hemorrhages extended to the peripheral edge of the retina while 37 percent were located in the posterior pole and peripapillary areas. For the non-AHT patients, 91 percent of hemorrhages were confined to the posterior pole and peripapillary, while only nine percent of the retinal hemorrhages extended to the periphery. The meta-analysis concludes that retinal hemorrhages “were more commonly bilateral, multilayered, and extending to the periphery in the AHT group. Non-AHT as a cause of retinal hemorrhage was very rare, but when present, hemorrhages were frequently unilateral, few, and restricted to the posterior pole.”¹⁹¹

A 2010 system review of studies by Bhardwaj confirmed that retinal hemorrhages are extremely specific for AHT, especially when the hemorrhages are bilateral, extensive, and multilayered.¹⁹² Bhardwaj adhered to clearly defined criteria for the inclusion and exclusion of studies, and eventually evaluated 560 AHT cases in 20 clinical studies.¹⁹³ Bhardwaj found that retinal hemorrhages had a specificity of 94 percent for AHT, which increased with moderate to severe, bilateral, pre-retinal, peripheral RH. The study also found that optic nerve sheath hemorrhages are more commonly found in AHT than in other conditions, and that traumatic retinoschisis and perimacular folds are seldom seen in non-AHT cases.¹⁹⁴

In the 2009 study by Vinchon described earlier in this article, ophthalmologists performed fundoscopic evaluations on 44 inflicted head injury (IHI) and 35 accidental trauma (AT) cases. RH were “rated as absent, mild, and severe, according to the depth and extent of the RH.”¹⁹⁵ Severe RH was the variable with the highest predictive value (.961) and, in the absence of direct ocular impact, severe RH was 100 percent specific for inflicted head trauma.¹⁹⁶ Severe RH

¹⁹⁰ Brauser, Deborah, *Retinal Findings Differ Between Abusive and Accidental Head Trauma in Children*, Medscape Medical News (May 3, 2010), viewed at <http://www.medscape.com/viewarticle/721141>.

¹⁹¹ *Id.*

¹⁹² Bhardwaj, G., et al., *A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma*, 117 *Ophthalmology* 983 (2010).

¹⁹³ *Id.* at 983.

¹⁹⁴ *Id.*

¹⁹⁵ *Id.* at 637.

¹⁹⁶ *Id.* at 640.

was found in 56.8 percent of IHI cases but in only AT case.¹⁹⁷ RH were only found in six AT cases, and were mild in five cases. The only AT case with severe RH had suffered facial trauma that suggested impact to the globe.¹⁹⁸ Vinchon concluded “that severe RH in the absence of facial trauma are specific of IHI.”¹⁹⁹

Multiple studies have shown that the incidence of any retinal hemorrhage after accidental head trauma is usually less than three percent with most studies finding no RH after accidental head injury.²⁰⁰ One study found an incidence of ten percent in accidents but there was a statistically significant difference in the distribution from abuse with those seen in accidents being much less severe.²⁰¹ In 2009, Dr. Binenbaum was the lead author on a study that concluded that “[r]etinal hemorrhages are highly associated with abusive head trauma, particularly in children younger than six months of age. Increasing retinal hemorrhage severity is correlated with increasing likelihood of abuse.”²⁰²

In very rare instances, severe fatal motor vehicle accidents²⁰³ and fatal crush injuries²⁰⁴ can be associated with diffuse, multilayered, bilateral retinal hemorrhages. However, a 2002 study found that retinal hemorrhages are uncommon in motor vehicle accidents.²⁰⁵ Defense experts will often cite a single case reported by Patrick Lantz which documented bilateral severe retinal hemorrhages in a 14-month-old child whose head was fatally crushed by a television,²⁰⁶ and a case reported by Lueder, in which a child also suffered a fatal head crush injury and severe bilateral retinal hemorrhages.²⁰⁷ However, Lueder himself wrote in a 2006 letter to the editor, “[a]lthough a small number of dot or blot hemorrhages may be seen occasionally in children with accidental head injury, the presence of diffuse hemorrhages almost always indicates nonaccidental injury.”²⁰⁸ A larger study of children with head crush injury did not show similar reti-

¹⁹⁷ *Id.* at 640.

¹⁹⁸ *Id.*

¹⁹⁹ *Id.* at 644.

²⁰⁰ Levin, Alex, *Retinal Hemorrhages: Advances in Understanding*, 56 *Pediatric Clinics N. Am.* 333, 338 (2009).

²⁰¹ Bechtel, Kirsten, Stoessel, Kathleen, and Leventhal, John M. et al., *Characteristics that Distinguish Accidental from Abusive Injury in Hospitalized Young Children with Head Trauma*, 114 *Pediatrics* 165, 167 (2004).

²⁰² Binenbaum, Gil M.D. et al., *Odds of Abuse Associated with Retinal Hemorrhages in Children Suspected of Child Abuse*, 13 *J. Am. Ass’n. Pediatric Ophthalmology Strabismus* 268 (2009).

²⁰³ Kivlin, Jane D., et al., *Retinal Hemorrhages in Children Following Fatal Motor Vehicle Crashes: A Case Series*, 126 *Archives Ophthalmology* 800 (2008).

²⁰⁴ Lantz, Patrick, *Perimacular Retinal Folds from Childhood Head Trauma*, 328 *Brit. Med. J.* 754 (2004); Lueder, Gregg T., M.D., Turner, Jane W., M.D. Ph.D., and Paschall, Robert, M.D., *Perimacular Retinal Folds Simulating Nonaccidental Injury in an Infant*, 124 *Archives Ophthalmology* 1782 (2006).

²⁰⁵ Vinchon, Matthieu et al., *Infantile Subdural Hematoma Due to Traffic Accidents*, 37 *Pediatric Neurosurgery*, 245 (2002).

²⁰⁶ Lantz, Patrick, *Perimacular Retinal Folds from Childhood Head Trauma*, 328 *Brit. Med. J.* 754 (2004).

²⁰⁷ Lueder, Gregg T., M.D., Turner, Jane W., M.D. Ph.D., and Paschall, Robert, M.D., *Perimacular Retinal Folds Simulating Nonaccidental Injury in an Infant*, 124 *Archives Ophthalmology* 1782 (2006).

²⁰⁸ Lueder, Gregg, *Retinal Hemorrhages in Accidental and Nonaccidental Injury*, 115 *Pediatrics* 192 (2005).

nal findings.²⁰⁹ The two crush cases documented by Lantz and Leuder represent unusual outliers, which although interesting, offer little relevance to abusive head injury.²¹⁰ The study by Bhardwaj found that unless a child was the victim of a motor vehicle accident or a high fall or crush injury that produced multiple skull fractures, the positive predictive value for AHT in a child with retinoschisis or retinal folds is 100 percent.²¹¹ The unique findings and circumstances of a motor vehicle accident or fatal head crush injury would allow a physician to easily distinguish these patients from those who suffered abusive head trauma.

Accurate Science: *Recent animal studies indicate that acceleration-deceleration forces can cause retinal hemorrhages in infants.*

“[T]hat in a clearly fatal shaking situation . . . no eye findings were produced . . . casts doubt on the ability of shaking to cause primary eye findings by the rotational mechanism.”²¹²

In the above quote, Dr. John Galaznik is discussing a study of three naturally occurring events in which two kittens and a baby rabbit were fatally shaken by a dog.²¹³ However, the authors of the referenced study note that a rabbit has smaller eyes which require much more force to injure in comparison to those of a human infant.²¹⁴ Also, human infants have large heads and weak neck muscles, and “the feline head and neck are better equipped to sustain acceleration–deceleration forces without injury.”²¹⁵ In addition, the mechanism of shaking is very different when an animal grabs its victim by the back of the neck.

Dr. Galaznik frequently references a 2007 abstract authored by Dr. Binenbaum in which piglets exposed to a single shake failed to manifest retinal hemorrhages.²¹⁶ This study only as-

²⁰⁹ Gnanaraj, L., et al., *Ocular Manifestations of Crush Head Injury in Children*, 21 Eye 5 (2007).

²¹⁰ Levin, A.V., *Retinal Hemorrhages of Crush Head Injury: Learning from Outliers*, 124 Archives Ophthalmology 1773 (2006).

²¹¹ Brown, S., *Abusive Head Trauma* (Letter to the Editor), 118 Ophthalmology 430-431 (2011).

²¹² Testimony of John Galaznik at 105, *State v. Daniel*, No. 05-CR-202-EV (Ga. Ct. Super. 2007). (Available through NCPCA).

²¹³ Brown, S., et al., *Natural Animal Shaking: A Model for Inflicted Neurotrauma in Children?* 11 J. Am. Ass’n. Pediatric Ophthalmology Strabismus 85 (2007).

²¹⁴ *Id.* at 2.

²¹⁵ *Id.*

²¹⁶ Testimony of Dr. John Galaznik at 106, *State v. Daniel*, No. 05-CR-202-EV (Ga. Ct. Super. 2007). (Available through NCPCA). (Discussing Gil Binenbaum et al., *An Animal Model to Study Retinal Hemorrhages in a Non-Impact Brain Injury*, 11 J. of Am. Ass’n. for Pediatric Ophthalmology and Strabismus 84 (2007)); see also, Testimony of John Galaznik at 1530-1531, *State v. Kennedy* (Wa. Ct. App. August 16, 2007) (discussing Binenbaum 2007). (Available through NCPCA).

sessed only a single shake along the axial plane, in contrast to the repetitive shaking in various directions and hypoxic damage that occurs in abusive head trauma.²¹⁷ Dr. Alex Levin, in a review of the Binenbaum study, cautioned “readers should not in any way interpret this abstract as indicating that shaking does not cause retinal hemorrhages.”²¹⁸ In fact, Binenbaum subsequently coauthored a study with Dr. Brittany Coats, using more thorough methodology, in which piglets were subjected to a single shake in one of three directions.²¹⁹ In this 2010 study, 73 percent of piglets were found to have retinal hemorrhages after a single acceleration-deceleration, with 51 percent of the piglets having bilateral RH.²²⁰

It is notable that 70 percent of the intraocular hemorrhages discovered in the 2010 Coats study were at the retinal periphery.²²¹ Statistically, peripheral hemorrhages are seen significantly more in abusive head injury when compared to accidental head trauma.²²² Although the authors rightly caution about extrapolating from piglets to human infants, the location of the retinal hemorrhages in the 2010 Coats study strengthens the hypothesis that retinal hemorrhages near the vitreous base result from traction on the retina by the vitreous as it is subjected to repeated acceleration-deceleration forces.

Accurate Science: *Retinal hemorrhages are not caused by CPR or bleeding disorders.*

Although the defense may proffer that the increased intrathoracic pressure from CPR could cause retinal hemorrhages in children, this contention is not borne out by the literature. The defense witness may discuss Purtscher syndrome, a condition seen in adults, but rarely in children, who suffer crushing chest injuries.²²³ Purtscher syndrome is defined by white polygonal retinal patches that are only rarely seen in AHT and have never been seen following CPR.²²⁴ A study by Morad that failed to substantiate a link between rib fractures and retinal hemorrhages further indicates that in-

²¹⁷ Binenbaum, G. et al., *An Animal Model to Study Retinal Hemorrhages in a Non-Impact Brain Injury*, 11 J. Am. Ass’n. Pediatric Ophthalmology Strabismus 84 (2007).

²¹⁸ Quarterly Update, Vol. XV, No. 1, p. 9. <http://www.quarterlyupdate.org>.

²¹⁹ Coats, Brittany et al., *Ocular Hemorrhages in Neonatal Porcine Eyes from Single, Rapid Rotational Events*, 51 Investigative Ophthalmology Visual Sci. 4792 (2010).

²²⁰ *Id.* at 4793.

²²¹ *Id.* at

²²² Bechtel, Kirsten, Stoessel, Kathleen, and Leventhal, John M. et al., *Characteristics that Distinguish Accidental from Abusive Injury in Hospitalized Young Children with Head Trauma*, 114 Pediatrics 165, 167 (2004).

²²³ Levin, Alex, *Retinal Hemorrhages: Advances in Understanding*, 56 Pediatric Clinics N. Am. 333, 337 (2009).

²²⁴ *Id.*

creased intrathoracic pressure does not cause retinal hemorrhages.²²⁵

Likewise, studies have failed to substantiate that CPR can cause retinal hemorrhages, particularly of the type and number associated with acceleration-deceleration injuries. In a 2002 study, 70 of 169 children who were given CPR prior to death had retinal hemorrhages, but in all 70 cases the retinal hemorrhages could be attributed to the child's cause of death, most often a head injury.²²⁶ A study in 1986 found six cases of retinal hemorrhages out of 54 patients who received CPR and survived.²²⁷ Of these six cases, four children were determined to have been abused, one was hit by a car, and the other had arterial hypertension and seizures.²²⁸

A 1997 study by Odom found a strong disassociation between CPR and RH, even in patients who also had a bleeding disorder.²²⁹ Odom prospectively studied 43 pediatric patients diagnosed with a non-traumatic, non-inflicted illness who survived after CPR, and found only a single case of RH.²³⁰ Odom's study was in a controlled setting—the pediatric ICU—and all 43 patients suffered the events precipitating cardiopulmonary arrest in the intensive care unit, eliminating the possibility of physical abuse as an etiology. The mean duration of chest compressions was 16 minutes, with 58 percent lasting between one and ten minutes. Five patients had chest compressions lasting over 40 minutes, and two patients had open chest cardiac massage. All of the patients survived their resuscitative efforts. Odom found small, punctate retinal hemorrhages—dissimilar to the diffuse RH in AHT cases—in only one patient.²³¹

In Odom's study, retinal hemorrhages were an extremely rare finding even in patients with a predisposition towards bleeding. Twenty-seven of the patients Odom studied had coagulopathy—a defect in the body's clotting process that can cause heavy bleeding after an injury—as indicated by an elevated prothrombin time (PT) or partial thromboplastin time (PTT). The PT and PTT are measures of how long it takes blood to clot, and an elevated PT or PTT is an indicator of a coagulopathy. Twenty patients had thrombocytopenia as indicated by a low platelet count, which can also cause abnormal bleeding. Eighteen patients were concurrently thrombocytopenic (low platelets) and had a coagulopathy (prolonged PT or PTT value). Despite this

²²⁵ Morad, Y. Avni, I. Capra, L. et al., *Shaken Baby Syndrome Without Intracranial Hemorrhage on Initial Computed Tomography*, 8 J. Am. Ass'n. Pediatric Ophthalmology Strabismus 521 (2004).

²²⁶ Gilleland, M., Luckenbach, M., *Are Retinal Hemorrhages Found after Resuscitation Attempts? A Study of the Eyes of 169 Children*, 14 Am. Forensic Med. Pathology 187 (1993).

²²⁷ Kanter, R., *Retinal Hemorrhage After Cardiopulmonary Resuscitation or Child Abuse*, 180 J. Pediatrics 430 (1986).

²²⁸ *Id.*

²²⁹ Odom, Amy et al., *Prevalence of Retinal Hemorrhages in Pediatric Patients After In-hospital Cardiopulmonary Resuscitation: A Prospective Study*, 99 Pediatrics 3 (1997).

²³⁰ *Id.* at 3.

²³¹ *Id.*

predisposition to bleeding, Odom found only one patient with small punctate retinal hemorrhages. Odom concluded that “retinal hemorrhages are a rare finding after chest compressions in patients with a coagulopathy or a low platelet count and are atypical of retinal hemorrhages seen in shaken baby syndrome.”²³²

Accurate Science: *Increased intracranial pressure (ICP) does not cause the diffuse retinal hemorrhages commonly associated with AHT.*

Despite a paucity of clinical evidence, defense experts will frequently testify that diffuse, multilayered retinal hemorrhages may be the result of increased intracranial pressure rather than inflicted injuries. Dr. John Galaznik testified in 2007 that “[W]hen faced with retinal hemorrhages extending in all four quadrants to the far periphery, one should consider venous occlusive disease as the etiology and an increased intracranial pressure does produce venous occlusion by its very presence.”²³³

Defense witnesses will sometimes point to adult cases of Terson syndrome, in which intracranial hemorrhage may be accompanied by retinal hemorrhages. Increased ICP or blood transmitted down the optic nerve sheath are possible causes of Terson Syndrome. One of the prominent findings associated with Terson’s syndrome in adults is increased intracranial pressure. However, Terson syndrome is rarely seen in children.²³⁴ In Morad’s study of children who were confirmed victims of AHT, only 5 percent of children showed swelling of the optic nerve (the cardinal sign of increased intracranial pressure) and there was no correlation between increased ICP and retinal findings.²³⁵

Studies of accidental injuries resulting in intracranial hemorrhages do not support the link between increased ICP and retinal hemorrhages. A study by Schloff of 57 non-abused children hospitalized for intracranial hemorrhages revealed retinal hemorrhages in only two patients.²³⁶ Of the 57 children studied, 27 were injured in known accidental trauma (motor vehicle accidents

²³² *Id.*

²³³ Testimony of John Galaznik at 1525, *State v. Kennedy* (Wa. Ct. App. August 16, 2007) (Available through NCPCA). *WA v. Andrew Kennedy*, (Wash., August 16, 2007).

²³⁴ Susan Schloff, M.D. et al., *Retinal Findings in Children with Intracranial Hemorrhage*, 109 *Ophthalmology* 1472, 1474 (2002).

²³⁵ Morad, Y., Avni, I., Capra, L. et al., *Shaken Baby Syndrome Without Intracranial Hemorrhage on Initial Computed Tomography*, 8 *J. of Am. Ass’n. Pediatric Ophthalmology Strabismus* 521 (2004).

²³⁶ Susan Schloff, M.D. et al., *Retinal Findings in Children with Intracranial Hemorrhage*, 109 *Ophthalmology* 1472, 1472-1474 (2002).

(MVA), sports accidents, falls, etc.), 24 were injured in surgeries, and six were injuries through other causes such as vessel malformations or infections. Fifty-five of the 57 children (96 percent) had no evidence of RH. One child had a single dot hemorrhage associated with presumed infection. The other child was involved in a MVA and had three flame and two deeper dot hemorrhages. No child had the severe or multi-layered retinal hemorrhages typical of AHT cases.

Thus it does not appear that either increased ICP or intracranial hemorrhages are likely to cause retinal hemorrhages in children. In the uncommon case that increased ICP does cause retinal hemorrhages, the hemorrhages would be few in number, small, pre- and/or intraretinal, and located near the optic nerve head, as opposed to the multilayered diffuse hemorrhages extending to the periphery typical of AHT cases.

Conclusion

This article provides an overview of some, but not all, of the issues likely to be raised by defense experts in AHT cases. For further analysis regarding defense expert testimony and cross-examination, please contact the National District Attorneys Association directly.

Page 43: **Blank**

Page 44: **Blank**

In 1985, the National District Attorneys Association recognized the unique challenges of criminals involving child victims and established the National Center for Prosecution of Child Abuse (NCPCA). NCPCA's mission is to reduce the number of children victimized and exploited by assisting prosecutors and allied professionals laboring on behalf of victims too small, scared or weak to protect themselves. NCPCA is a voice for the voiceless. NCPCA is dedicated to saving the lives of children who cannot defend themselves.

What Can NDAA's National Center for Prosecution of Child Abuse Do For You?

National Conferences and Trial Advocacy Trainings As the nation's most prominent and respected trainer on issues related to child maltreatment, NCPCA sponsors a series of multidisciplinary national conferences and advanced trial advocacy courses for prosecutors, law enforcement, medical and mental health professionals, forensic interviewers, social workers, child advocates and allied professionals.

Customized State and Local Trainings

NCPCA's senior attorneys with expertise in child maltreatment are regularly solicited to teach at international, national, regional, state and local child abuse conferences.

Free Online Training

Recently, NCPCA created an E-Learning Center. The Center houses free online trainings regarding the best practices and procedures used in investigating and prosecuting child abuse cases. Currently, six trainings which focus on children testifying in court are available on the site. Soon new trainings in the form of audio podcasts, full length courses, and webinars will also be available.

Technical Assistance Requests

Each year, NCPCA receives thousands of requests from prosecutors and other allied professionals seeking assistance related to specific child abuse cases. NCPCA offers immediate assistance by providing substantive legal, medical and psychological research, deciphering complex medical or forensic evidence, brainstorming trial strategies, expert witness assistance, legislative review and statutory analysis.

Publications

NCPCA publishes the monthly substantive newsletter Update, the Child Sexual Exploitation Update, and the web-based Update Express. NCPCA also publishes the two-volume manual Investigation and Prosecution of Child Abuse, 3rd edition. This book was originally published in 1987 and quickly became the authoritative text for handling criminal cases in child abuse. A new manual with updated material including child protection, commercial sexual exploitation and domestic minor sex trafficking will be available in 2013.



National District Attorneys Association

44 Canal Center Plaza, Suite 110, Alexandria, VA 22314

www.ndaa.org